ACTA COLL MED PHILAD INSTIT A D M DCCLXXVII

Digitized by the Internet Archive
in 2013

http://archive.org/details/transactionsofco3531coll

TRANSACTIONS

OF THE

COLLEGE OF PHYSICIANS

OF

PHILADELPHIA

THIRD SERIES

VOLUME THE FIFTY-THIRD

PHILADELPHIA
PRINTED FOR THE COLLEGE
1931

NOTICE

The present volume of TRANSACTIONS contains the papers read before the College from January, 1931, to December, 1931, inclusive.

The Committee of Publication thinks it proper to say that the College holds itself in no way responsible for the statements, reasonings, or opinions set forth in the various papers published in its TRANSACTIONS.

EDITED BY

WALTER G. ELMER, M.D.

"This volume is published in part by the income from the Francis Houston Wyeth Fund."

DORNAN, PRINTER

CONTENTS.

List of Presidents of the College vii
List of Officers and Standing Committees viii
List of Fellows of the College xi
List of Associate Fellows of the College lvii
Necrological List for 1931 lix
List of Prizes and Lectures lx
Memoir of Joseph Seal Neff, M.D. lxv

Annual Address of the President. By Francis R. Packard, M.D. . . 1
Symposium: The Therapeutic Use and Abuse of Fluids. Introduction. Purpose of the Symposium. By J. Harold Austin, M.D. . 8
Intravenous Therapy in Dehydration and Toxemia. By J. Harold Austin, M.D., and I. S. Raydin, M.D. 10
Nonspecific Protein Therapy. Some Cautions as to Its Use. By Richard A. Kern, M.D. 17
Nonspecific Protein Therapy in Surgical Conditions. By Lee Rademaker, M.D. 21
Discussion from a Surgical Standpoint. By John H. Gibbon, M.D. 25
Discussion from a Surgical Standpoint. By Damon B. Pfeiffer, M.D. 27
Sympathetic Activity in Certain Diseases, Especially Those of the Peripheral Circulation. The Mary Scott Newbold Lecture. Lecture XXV. By W. J. Merle Scott, M.D., and John J. Morton, M.D. . . . 30
The Community Health Program. By C.-E. A. Winslow, Dr. P.H. . 66
Symposium. The Past Five Years of Syphilologic Progress:
Recent Experimental Contributions to Syphilis. By Joseph V. Klauder, M.D. 92
Recent Developments in the Serology of Syphilis. By John A. Kolmer, M.D. 97
Bismuth in the Treatment of Syphilis. By Carroll S. Wright, M.D. 101
Fever Therapy in the Treatment of Syphilis. By Jay Frank Schamberg, M.D. 108
The Newer Arsenicals in the Treatment of Syphilis. Therapeutic Use and Control of Reactions. By Arthur G. Schoch, M.D. . 113
The Newer Conceptions of the Diagnosis and Treatment of Early Syphilis. By Vaughn C. Garner, M.D. 118
The Prevention and Treatment of Prenatal Syphilis. By Donald M. Pillsbury, M.D. 122
Syphilis as a Problem in Preventive Medicine. By John H. Stokes, M.D. 127

Vasovagal Syncope and the Carotid Sinus Mechanism, with Comments on Gowers' and Nathnagel's Syndrome. The Mary Scott Newbold Lecture. Lecture XXVII. By Sir Thomas Lewis, M.D., F.R.S. . 132

Osteoarthritis Deformans Contrasted with Osteoarthritis Deformans Juvenilis. The Mary Scott Newbold Lecture. Lecture XXVIII. By Prof. F. J. Lang 144

How London and Edinburgh Influenced Medicine in Philadelphia in the Eighteenth Century. The S. Weir Mitchell Oration. By Francis R. Packard, M.D. 151

Diverticulosis of the Alimentary and Urinary Tracts. The Mütter Lecture. By Edwin Beer, M.D. 182

Medical Science in the Service of the State: Some Professional Experiences. The Mary Scott Newbold Lecture XXVI. By George Burgess Magrath, A.M., M.D. 200

Proceedings of the Section on Ophthalmology

Report of a Case of Reconstruction of Eye Socket. By George H. Cross, M.D. 203

Large Penetrating Wound of Lower Lid and Eyeball. By Frederick Krauss, M.D. 203

Homonymous Hemianopsia in Diabetes. By George E. deSchweinitz, M.D. 205

Cysts of the Orbit. By Frederick Krauss, M.D. 205

The Treatment of Interstitial Keratitis. By Joseph V. Klauder, M.D., and Harold F. Robertson, M.D. 206

Unsuspected Brawny Scleritis in a Case of Retinal Detachment with Secondary Glaucoma. By T. B. Holloway, M.D., and W. E. Fry, M.D. 208

Congenital Cataract With Unusual Visual Acuity. By William Zentmayer, M.D. 209

Case of Spontaneous Cyst of the Iris. By Edward A. Shumway, M.D. 211

Melanosis of Anterior Segment of Globe With Elevations on Iris. By H. Maxwell Langdon, M.D. 212

A Note on Radiational Cataract. By George E. deSchweinitz, M.D., and B. F. Baer, Jr., M.D. 214

Localizing Tears in Retinal Detachment. By Alfred Cowan, M.D., and L. F. McAndrews, M.D. 216

A Case of Bitemporal Paracentral Scotoma. By C. E. G. Shannon, M.D. 217

Nonglaucomatous Halos. By John T. Carpenter, M.D., and Chapin Carpenter, M.D. 219

The Hazards of Antifreeze Methanol. By Max Trumper, Ph.D. . . 220

Spontaneous Complete Absorption of a Cataractous Lens. By T. B. Holloway, M.D., and Alfred Cowan, M.D. 222

Senile Macular Changes of the Retina Associated With Focal Infection. By Hunter Scarlett, M.D. 226

The Syphilitic Optic Atrophies. By Joseph Earle Moore, M.D. . . . 226

Blepharoplastic and Ptosis Operative Procedure. By Vilray P. Blair, M.D. 228

Left Plastic Iritis Which Does Not Respond to Mydriatic and Parenteral Protein Injections. By William F. Bonner, M.D. 228

A Case of Bilateral Optic Atrophy Due to Methyl Alcohol Poisoning. By C. E. G. Shannon, M.D. 229

Removal of Oxidized Tissue Remaining After Foreign Body of Cornea (A Handle for Corneal Burrs). By George H. Cross, M.D. 231

Lupus Erythematosus of the Conjunctiva and Eyelids. By Joseph V. Klauder, M.D., and Perce De Long, M.D. 232

Lymphomas of the Conjunctiva. By C. E. G. Shannon, M.D., and L. F. McAndrews, M.D. 233

The Recession Operation in Squint. By Luther C. Peter, M.D. . . . 234

The Ophthalmological Importance of Focal Infective Prostatitis. By P. S. Pelouze, M.D. 236

Sarcoma of the Choroid. By T. B. Holloway, M.D. 238

Proceedings of the Section on Otology and Laryngology

Roentgenographic Anatomy and Physiology of the Larynx and Adjacent Structures. By Henry K. Pancoast, M.D. 239

The Value of Roentgen Diagnosis in Diseases of the Neck and Upper Respiratory Tract. (Lantern Slide Demonstration.) By Eugene P. Pendergrass, M.D. 240

How Dr. G. Hudson Makuen Treated Stammering. By Mrs. Mary S. Steel (by invitation) 243

Experimental Proof that Voice Cannot be Maintained at a Level. By Douglas MacFarlan, M.D. 246

Lesions of the Pterygomaxillary Fossa (Report of Three Cases). By Robert J. Hunter, M.D. 247

Postoperative Treatment by Radium in Radical Mastoidectomy. By Joseph C. Keeler, M.D. 251

The Usual Course and Relations of the Nervus Facialis and the Practically Important Variations. By O. V. Batson, M.D. 252

Early Intrinsic Cancer of the Larynx—Diagnosis and Treatment. By Gabriel Tucker, M.D. 254

A Modified Attic Drainage in Chronic Suppurative Otitis Media. Preliminary Report. By James A. Babbitt, M.D. 258

Malignant Conditions of the Laryngopharynx. By Henry B. Orton, M.D. 261

Joint Meeting, Sections on Otology and Laryngology; Public Health and Industrial Medicine. Audiometric Findings and Clinical Records in Philadelphia Public Schools. By Walter S. Cornell, M.D., Marie Currie Frey, M.D., Miss Roberta Laird 263

Noise in Industry, Its Effect on the Hearing and on General Health. By Henry Field Smyth, M.D. 264

Indications for Removal of Tonsils and Adenoids. By George B. Wood, M.D. 268

Cerebrospinal Rhinorrhea (Report of Two Cases). By J. C. Donnelly, M.D. 271

Serious Complications Following Tonsillectomy (Case Reports). By George B. Wood, M.D. 274

Osteoma of the Paranasal Sinuses (Case Report). By J. Austin Smith, M.D. 278

A Short Résumé of the Results of the Ballance-Duel Research in Nerve Anastomosis and the Employment of Nerve Grafts in the Surgical Treatment of Facial Palsy (Moving Pictures and Lantern Slides). By Arthur B. Duel, M.D. 279

Motion Pictures of the Larynx. By C. A. Heatley, M.D. 284

Motion Pictures of Radical Frontal Sinus Operations. By Samuel Skillern, M.D. 286

Motion Picture Demonstration of Radical Maxillary Sinus Operation Under Local Anesthesia (Caldwell-Luc). By Henry Dintenfass, M.D. 288

Motion Picture Demonstration of Obstructive Dyspnea: Bronchoscopic Observations. By Gabriel Tucker, M.D. 289

Proceedings of the Section on General Medicine 292

Proceedings of the Section on Medical History 295

Annual Report of the Library Committee 297

LIST

OF THE

PRESIDENTS OF THE COLLEGE FROM THE TIME OF ITS INSTITUTION

ELECTED	
1787	JOHN REDMAN
1805	WILLIAM SHIPPEN
1809	ADAM KUHN
1818	THOMAS PARKE
1835	THOMAS C. JAMES*
1835	THOMAS T. HEWSON
1848	GEORGE B. WOOD
1879	W. S. W. RUSCHENBERGE
1883	ALFRED STILLÉ
1884	SAMUEL LEWIS†
1884	J. M. DA COSTA
1886	S. WEIR MITCHELL
1889	D. HAYES AGNEW
1892	S. WEIR MITCHELL
1895	J. M. DA COSTA
1898	JOHN ASHHURST, JR.
1900	W. W. KEEN
1902	HORATIO C. WOOD
1904	ARTHUR V. MEIGS
1907	JAMES TYSON
1910	GEORGE E. DE SCHWEINITZ
1913	JAMES CORNELIUS WILSON
1916	RICHARD H. HARTE
1919	WILLIAM J. TAYLOR
1922	THOMAS R. NEILSON
1925	HOBART A. HARE
1928	JOHN H. GIBBON
1931	FRANCIS R. PACKARD

* Died four months after his election.
† Resigned on account of ill-health.

COLLEGE OF PHYSICIANS OF PHILADELPHIA

1931

OFFICERS AND STANDING COMMITTEES

PRESIDENT

FRANCIS R. PACKARD, M.D.

VICE-PRESIDENT

ALFRED STENGEL, M.D.

CENSORS

GEORGE E. DE SCHWEINITZ, M.D.
JAMES C. WILSON, M.D.
WILLIAM J. TAYLOR, M.D.
THOMAS R. NEILSON, M.D.

SECRETARY

JOHN H. GIRVIN, M.D.

TREASURER

CHARLES F. MITCHELL, M.D.

HONORARY LIBRARIAN

ASTLEY P. C. ASHHURST, M.D.

COUNCILLORS

To serve until January, 1932

JOHN B. CARSON, M.D.
EDWARD B. HODGE, M.D.

To serve until January, 1933

WILLIAM D. STROUD, M.D.
HERMAN B. ALLYN, M.D.

To serve until January, 1934

EDWARD J. KLOPP, M.D.
GEORGE M. PIERSOL, M.D.

COMMITTEE OF PUBLICATION

THOMPSON S. WESTCOTT, M.D., *Ch'n*
WALTER G. ELMER, M.D.
ARTHUR H. HOPKINS, M.D.

LIBRARY COMMITTEE

GEORGE W. NORRIS, M.D., *Ch'n*
ALBERT P. BRUBAKER, M.D.
EDWARD B. KRUMBHAAR, M.D.
JOHN H. JOPSON, M.D.
H. R. M. LANDIS, M.D.
The HONORARY LIBRARIAN *ex-officio*

COMMITTEE OF MÜTTER MUSEUM

GEORGE P. MULLER, M.D., *Ch'n*
MILTON J. GREENMAN, M.D.
HERBERT FOX, M.D.

HALL COMMITTEE

E. HOLLINGSWORTH SITER, M.D., *Ch'n*
B. ALEX. RANDALL, M.D.
ELLISTON J. MORRIS, M.D.
GEORGE W. OUTERBRIDGE, M.D.
PHILIP F. WILLIAMS, M.D.

COMMITTEE ON DIRECTORY FOR NURSES

FREDERICK FRALEY, M.D., *Ch'n* — HORACE H. JENKS, M.D.
HENRY P. BROWN, JR., M.D.

COMMITTEE ON FINANCE

WILLIAM J. TAYLOR, M.D., *Ch'n* — O. H. P. PEPPER, M.D.
DAVID RIESMAN, M.D. — The PRESIDENT and TREASURER, *ex-officio*

COMMITTEE ON ENTERTAINMENTS

NORRIS W. VAUX, M.D., *Ch'n* — HENRY P. BROWN, JR., M.D.
ARTHUR NEWLIN, M.D. — J. STEWART RODMAN, M.D.
The PRESIDENT, *ex-officio*

COMMITTEE ON SCIENTIFIC BUSINESS

W. ESTELL LEE, M.D., *Ch'n* — EUGENE L. OPIE, M.D.
DAMON B. PFEIFFER, M.D. — The PRESIDENT and SECRETARY, *ex-officio*

COMMITTEE ON PUBLIC HEALTH AND PREVENTIVE MEDICINE

ELMER H. FUNK, M.D., *Ch'n* — JAMES M. ANDERS, M.D.
HENRY FIELD SMYTH, M.D. — The PRESIDENT, *ex-officio*

COMMITTEE ON ALVARENGA PRIZE

RICHARD A. KERN, M.D., *Ch'n* — WILLIAM D. STROUD, M.D.
BAXTER L. CRAWFORD, M.D. — CLIFFORD B. LULL, M.D.
I. S. RAVDIN, M.D.

COMMITTEE ON NATHAN LEWIS HATFIELD PRIZE AND LECTURESHIP

(until February, 1934)

WILLIAM R. NICHOLSON, M.D., *Ch'n* — RALPH PEMBERTON, M.D.
JAMES A. BABBITT, M.D.

TRUSTEES OF NATHAN LEWIS HATFIELD PRIZE AND LECTURESHIP

R. N. DOWNS, JR., M.D. — HENRY K. DILLARD, JR., M.D.
ARTHUR H. GERHARD, M.D.

TRUSTEE UNDER THE WILL OF THOMAS SKELTON HARRISON

(until February, 1932)

FREDERICK FRALEY, M.D.

COMMITTEE ON WEIR MITCHELL ORATION

(until February, 1933)

G. E. DE SCHWEINITZ, M.D., *Ch'n* — FRANCIS W. SINKLER, M.D.
CHARLES H. FRAZIER, M.D. — ALBERT P. BRUBAKER, M.D.
CHARLES W. BURR, M.D.

COMMITTEE ON MARY SCOTT NEWBOLD LECTURES

WILLIAM J. TAYLOR, M.D., *Ch'n* — WILLIAMS B. CADWALADER, M.D.
EDWARD J. KLOPP, M.D.
The PRESIDENT and CHAIRMAN COMMITTEE ON SCIENTIFIC BUSINESS, *ex-officio*

SECTIONS

Ophthalmology—*Chairman*, H. Maxwell Langdon, M.D.
Clerk, A. G. Fewell, M.D.

Otolaryngology—*Chairman*, Ralph Butler, M.D.
Clerk, Karl M. Houser, M.D.

General Medicine—*Chairman*, Henry K. Mohler, M.D.
Clerk, Richard A. Kern, M.D.

Medical History—*Chairman*, Herman B. Allyn, M.D.
Clerk, E. J. G. Beardsley, M.D.

Public Health and Industrial Medicine—*Chairman*, Walter S. Cornell, M.D.
Clerk, Alex. Heron Davisson, M.D.

FELLOWS

OF THE

COLLEGE OF PHYSICIANS OF PHILADELPHIA

DECEMBER, 1931

* Non-resident Fellows. † Fellows who have commuted dues. ‡ Deceased.

ELECTED

1892. Abbott, Alexander Crever, M.D., Sc.D., Dr. P.H., 4016 Pine St.

1912. Addison, William H. F., A.B., M.D., Professor of Normal Histology and Embryology in the University of Pennsylvania. Medical Laboratories, University of Pennsylvania.

1926. Adler, Francis Heed, M.A., M.D., Assistant Surgeon, Wills Eye Hospital; Instructor in Ophthalmology and Physiology, University of Pennsylvania. 313 S. Seventeenth St.

1905. Adler, Lewis H., Jr., M.D., Formerly Professor of Proctology in the Graduate School of Medicine, University of Pennsylvania; formerly Prosector to the Professor of Anatomy in the University of Pennsylvania; Consulting Surgeon to the Charity Hospital. 313 S. Seventeenth St.

1914. Aiken, Thomas Gerald, M.D., Physician-in-Chief to the Chester County Hospital, West Chester, Pa.; Pathologist to the Country Branch of the Rush Hospital for Tuberculosis. Berwyn, Pa.

1906. Allen, Francis Olcott, Jr., A.B., M.D. 2216 Walnut St.

1896. Allyn, Herman B., A.B., M.D., Consulting Physician to the Philadelphia General Hospital. 3910 Chestnut St.

1888. Anders, James M., M.D., Ph.D., LL.D., Sc.D., Emeritus Professor of Medicine in the Graduate School of Medicine, University of Pennsylvania; Officer de l'Instruction Publique, Chevalier Legion of Honor. 250 S. Seventeenth St.

1905. Anspach, Brooke M., M.D., Professor of Gynecology in the Jefferson Medical College; Attending Gynecologist, Jefferson Hospital; Attending Gynecologist to the Bryn Mawr Hospital. 1827 Spruce St.

ELECTED

1930. Appel, Kenneth E., A.B., A.M., Ph.D., M.D., Assistant Professor of Psychiatry, Medical School, University of Pennsylvania; Chief of Out-Patient Department and Psychiatrist, Institute for Mental Hygiene, Pennsylvania Hospital; Chief of Neuro-Psychiatric Pay Clinic, Pennsylvania Hospital, Haverford, Pa.

1905. Appleman, Leighton F., M.D., Attending Surgeon to the Wills Eye Hospital; Associate Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Ophthalmologist to the Frederick Douglass Memorial Hospital and to the Burd School; Demonstrator of Pharmacy and Materia Medica, and Instructor in Therapeutics in the Jefferson Medical College. 308 S. Sixteenth St.

1922. Arnett, John Hancock, A.B., M.D., Chief of Medical Service "B," Episcopal Hospital; Instructor in Medicine, Medical School and Graduate School of Medicine, University of Pennsylvania. 2116 Pine St.

1906. Ashhurst, Astley Paston Cooper, A.B., M.D., Professor of Clinical Surgery, University of Pennsylvania; Surgeon to the Episcopal Hospital and to the Philadelphia Orthopædic Hospital and Infirmary for Nervous Diseases. N. W. Cor. Twenty-second and Chestnut Sts.

1893. Ashton, Thomas G., M.D. Wynnewood, Pa.

1920. Astley, George Mason, M.D., Assistant Surgeon, Samaritan Hospital. 5317 Master St.

1914. Austin, J. Harold, B.S. (Univ. of Penna.), M.D., Professor of Research Medicine, University of Pennsylvania. John Herr Musser Department of Research Medicine, University of Pennslyvania.

1906. Babbitt, James A., A.B. (Yale), A.M. (Haverford), M.D., Assistant Professor of Oto-laryngology, University of Pennsylvania; Associate Professor of Oto-laryngology, Graduate School of Medicine, University of Pennsylvania; Laryngologist and Aurist, Lankenau Hospital; Oto-laryngologist, Children's Hospital. 1912 Spruce St.

1910. Baer, Benjamin F., Jr., B.S., M.D., Associate Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Assistant Ophthalmologist, University Hospital; Attending Surgeon to Wills Eye Hospital. 1927 Spruce St.

1911. Baldwin, James Harvey, A.B., M.D., Surgeon to the Methodist Hospital. 1426 Pine St.

ELECTED

1916. Balentine, Percival L., M.D., Assistant Surgeon, Wills Eye Hospital. 407 Weightman Building, 1524 Chestnut St.

1898. Balliet, Tilghman M., A.M., M.D., Professor of Therapeutics at Dartmouth College, Hanover, N. H.; Physician to the Old Man's Home. 3920 Sansom St.

1911. Barnard, Everett P., M.D., Obstetrician to the Maternity Hospital; Assistant Instructor in Obstetrics in the University of Pennsylvania. 1820 S. Rittenhouse Square.

1924. Bartle, Henry J., B. S., M.D., Clinical Assistant, Department for Disease of the Stomach and Bowels, Jefferson Medical College and Hospital. 1930 Chestnut St.

1931. Bates, William, M.D. 2029 Pine St.

1921. Bauer, Edward L., M.D., Professor of Children's Diseases, Jefferson Medical College; Pediatrist to the Jefferson and Memorial Hospitals and the Philadelphia Hospital for Contagious Diseases; Consulting Pediatrist, Germantown Hospital. 345 S. Nineteenth St.

*1883. Baum, Charles, A.M., M.D., Ph.D. 123 W. Main St., Middletown, Pa.

1922. Bazett, Henry Cuthbert, M.A., M.D. (Oxon.), F.R.C.S. (England), Professor of Physiology, University of Pennsylvania. Bryn Mawr, Pa.

1908. Beardsley, Edward J. G., M.D., L.R.C.P. (Lond.), Clinical Professor of Medicine in the Jefferson Medical College; Chief Clinical Assistant in the Out-Patient Medical Department of the Jefferson Medical College Hospital; Assistant Physician to the Jefferson Hospital. 1919 Spruce St.

1930. Beardwood, Joseph T., A.B., M.D., Associate Physician, Presbyterian Hospital; Associate in Cardiology, Graduate School of Medicine, University of Pennsylvania, Physician in Chief, Department of Metabolic Diseases, Abington Memorial Hospital. 256 S. Twenty-first St.

1930. Belk, William Parks, A.B., M.D., Pathologist, Bryn Mawr Hospital; Senior Histologist, Philadelphia General Hospital. Bryn Mawr, Pa.

*1916. Bell, William Hemphill, M.D., Captain, Medical Corps, U. S. Navy, Commanding Officer, U. S. Naval Medical School. 1869 Wyoming Ave., N.W., Washington, D. C.

1923. Bergey, David H., A.M., M.D., B.S., Dr. P.H., Professor of Hygiene and Bacteriology, University of Pennsylvania. 206 S. Fifty-third St.

*1903. Biggs, Montgomery H., M.D., Surgeon to the Rutherford Hospital; Chief Surgeon of the Carolina, Clinchfield and Ohio Railway. Rutherfordton, N. C.

ELECTED

1918. Billings, Arthur E., M.D., Assistant Professor of Surgery, Jefferson Medical College; Assistant Surgeon, Jefferson Hospital; Surgeon to Pennsylvania and Bryn Mawr Hospitals. 2020 Spruce St.

1917. Birdsall, Joseph C , A.B., A.M., M.D., Associate Professor of Urology in the Graduate School of Medicine, and Urologist to the Hospital of the Graduate School of Medicine, University of Pennsylvania; Genito-urinary Surgeon, Presbyterian Hospital. 4610 Spruce St.

1908. Bland, Pascal Brooke, M.D., Professor of Obstetrics, Jefferson Medical College; Visiting Obstetrician, Jefferson Medical College; Gynecologist to St. Joseph's Hospital. 1621 Spruce Street.

1917. Block, Frank B., A.B., M.D., Surgeon to the Jewish Hospital. 1121 Medical Arts Building.

1894. Bochroch, Max H., M.D., Professor of Nervous and Mental Diseases, Temple University; Visiting Physician to the Psychopathic Wards of the Philadelphia General Hospital. 1539 Pine St.

1929. Bockus, Harry L., B.S., M.D., Professor of Gastroenterology, Graduate School of Medicine, University of Pennsylvania, and Attending Gastroenterologist, Graduate Hospital; Attending Physician, Presbyterian Hospital. 250 S. Eighteenth St.

1896. Boger, John A., A.M., M.D., Surgeon to St. Mary's Hospital; Surgeon to the Stetson Hospital. 2213 N. Broad St.

1928. Boles, Russell S., M.D., Associate in Medicine, University of Pennsylvania; Visiting Physician, Philadelphia General Hospital; Consulting Gastroenterologist, U. S. Veterans' Bureau, Philadelphia. Rittenhouse Plaza.

1923. Bond Earl, D., A.B., M.D., Physician-in-Chief, Pennsylvania Hospital, Department for Mental and Nervous Diseases; Professor of Psychiatry, School of Medicine and Graduate School of Medicine, University of Pennsylvania. Bryn Mawr, Pa.

1911. Bonney, Charles W., A.B., M.D., Assistant Professor of Topographic and Applied Anatomy in the Jefferson Medical College. 1117 Spruce St.

1924. Boon, David J., M.D., Opthalmologist, Memorial Hospital and Philadelphia Hospital for Contagious Diseases; Assistant Ophthalmic Surgeon, Episcopal Hospital. 1532 N. Fifteenth Street.

ELECTED

1931. Bortz, Edward L., A.B., M.D., Visiting Physician to Lankenau Hospital; Assistant Professor of Medicine, Graduate School of Medicine, University of Pennsylvania. 2021 W. Girard Ave.

1921. Borzell, Francis Frank, M.D., Roentgenologist, Frankford Hospital; Chief Clinical Assistant, Roentgen Department, Jefferson Medical College; *X*-Ray Consultant, State Hospital for the Insane, Norristown, Pa. 4940 Penn St.

‡1913. Boston, L. Napoleon, A.M., M.D., Professor of Physical Diagnosis, Graduate School of Medicine, University of Pennsylvania; Professor of Principles and Practice of Medicine and Clinical Medicine, Woman's Medical College. 1930 Chestnut St.

1928. Bothe, Albert Edward, B.S., M.D., M.S., Instructor in Surgery, Medical School and Instructor in Urology, Graduate School, University of Pennsylvania; Assistant in Urology, Presbyterian Hospital; Urologist, Misericordia Hospital. 133 S. Thirty-sixth St.

1928. Bothe, Frederick A., M.S., M.D., Assistant Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Assistant Surgeon, Presbyterian Hospital; Associate Surgeon, Children's Hospital. 133 S. Thirty-sixth St.

1919. Bowen, David Ralph, M.D., Radiologist to the Pennsylvania Hospital. 82 W. Lacrosse Ave., Lansdowne, Pa.

1923. Bower, John O., M.D., Surgeon to the Philadelphia General and Northeastern Hospitals; Consulting Surgeon to United States Veterans' Hospitals; Clinical Professor of Surgical Research, Temple University. 2008 Walnut St.

1891. Boyd, George, M., M.D., Professor of Obstetrics, Graduate School of Medicine, University of Pennsylvania; Consulting Obstetrician to the Preston Retreat and Philadelphia General Hospital. 1909 Spruce St.

1907. Boyer, Henry Percival, M.D., Neurologist to the Stetson Hospital; Physician to the Philadelphia Home for Incurables; Medical Electrician to the Orthopedic Hospital and Infirmary for Nervous Diseases. 4602 Baltimore Ave.

1925. Boykin, Irvine M., M.D., Associate Surgeon to the Episcopal Hospital; Associate in Surgery, Graduate School of Medicine, Instructor in Surgery, School of Medicine, University of Pennsylvania. 922 Medical Arts Building.

1929. Bradbury, Samuel, M.D., Medical Director, Out-Patient Department, Pennsylvania Hospital; Chief of Service, "B," Department of Medicine, Germantown Dispensary and Hospital. 151 W. Coulter St.

ELECTED

1907. Bradley, William N., Ph.G., M.D., Visiting Pediatrist to Philadelphia General Hospital; Associate in Pediatrics, School of Medicine, University of Pennsylvania; Associate Professor of Pediatrics, Graduate School of Medicine, University of Pennsylvania. 1725 Pine St.

1921. Bransfield, John W., M.D., Clinical Professor of Surgery, Woman's Medical College; Visiting Surgeon to St. Agnes, St. Vincent's and Woman's College Hospitals. 2101 Spruce Street.

1903. Branson, Thomas F., M.D., Attending Physician to the Bryn Mawr Hospital. Rosemont, Pa.

1900. Brinton, Ward, A.M., M.D., Chief of Division of Tuberculosis, Department of Public Health of Philadelphia; Clinical Professor of Medicine, Woman's Medical College of Pennsylvania; Visiting Physician, Department for Tuberculosis, Philadelphia General Hospital, and Eaglesville Sanatorium. 1423 Spruce St.

1917. Bromer, Ralph S., A.B. (Yale), M.D., Associate in Radiology, Graduate School of Medicine, and Instructor in Roentgenology, School of Medicine, University of Pennslyvania; Roentgenologist to the Episcopal, the Orthopedic and the Children's Hospitals. 504 W. Mt. Airy Ave.

1919. Brown, Henry P. Jr., M.D., Surgeon and Chief of Out-Patient Clinic of the Pennsylvania Hospital; Surgeon to the Children's Hospital; Associate Surgeon, Presbyterian Hospital; Assistant Professor of Surgery in the Graduate School of Medicine, University of Pennsylvania. 1930 Chestnut St.

1916. Brown, Samuel Horton, M.D. 1930 Chestnut St.

1887. Brubaker, Albert P., A.M., M.D., Emeritus Professor of Physiology and Medical Jurisprudence in the Jefferson Medical College. 3426 Powelton Ave.

1930. Bucher, Carl J., B.S., M.D. The Westbury, Fifteenth and Spruce Sts.

1916. Buckley, Albert Coulson, M.D., Medical Superintendent, Friends' Hospital, Frankford; Professor of Psychiatry, Graduate School of Medicine, and Associate Professor of Psychiatry, School of Medicine, University of Pennsylvania; Neurologist to Frankford Hospital; Honorary Consultant to the Psychopathic Department, Philadelphia General Hospital. Frankford, Phila.

1928. Burden, Verne G., M.D., M.S., Surgeon to St. Joseph's and Philadelphia General Hospitals; Instructor in Surgery, Graduate School of Medicine, University of Pennsylvania. The Warwick, Seventeenth and Locust Sts.

ELECTED

1927. Burns, Michael A., M.D., Associate Professor Nervous and Mental Diseases, Jefferson Medical College; Chief of the Nervous and Mental Clinic, Jefferson Hospital; Visiting Neurologist, Philadelphia General Hospital; Neuropsychiatrist, St. Mary's Hospital. N. E. Cor. Twentieth and Chestnut Sts.

1906. Burns, Stillwell C., M.D., Associate Professor of Surgery in the Graduate School of Medicine, University of Pennsylvania. 1925 Spring Garden St.

1892. Burr, Charles W., M.D., Professor of Mental Diseases in the University of Pennsylvania; Neurologist to the Philadelphia General Hospital. 1918 Spruce St.

1906. Butler, Ralph, M.D., Professor of Laryngology in the Graduate School of Medicine, University of Pennsylvania; Laryngologist and Aurist to the Lankenau Hospital. 1930 Chestnut Street.

*1908. Cadbury, William W., A.M., M.D., Lingnan University, Canton, China.

†1907. Cadwalader, Williams B., M.D., Clinical Professor of Neurology, School of Medicine, University of Pennsylvania; Neurologist to the Presbyterian Hospital; Consulting Neurologist to the Bryn Mawr Hospital. 133 S. Thirty-sixth St.

1928. Campbell, Edward H., M.D., Assistant Oto-laryngologist, Pennsylvania and Children's Hospitals, and in Out-Patient Department of Lankenau Hospital; Instructor in Oto-laryngology in the Medical School and Graduate School of Medicine, University of Pennsylvania. 2117 Chestnut St.

1931. Cantarow, Abraham, M.D., Instructor in Medicine, Jefferson Medical College; in charge of Laboratory of Biochemistry, Jefferson Hospital; Assistant Physician, Philadelphia General Hospital. 2031 Pine St.

1931. Cariss, Walter L., A.B., M.D., Associate in Oto-laryngology, Presbyterian Hospital. 2043 Walnut St.

1905. Carmany, Harry S., M.D., Surgeon to Memorial Hospital, Roxborough; Associate Surgeon of the Episcopal Hospital. 366 Green Lane, Roxborough.

1910. Carnett, John Berton, M.D., Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Associate in Surgery, Medical Department, University of Pennsylvania; Surgeon to Philadelphia General Hospital; Chief Surgeon to the American Stomach Hospital. 2012 Spruce St.

ELECTED

1930. Carpenter, Chapin, A.B., M.D., Assistant Ophthalmologist, Bryn Mawr Hospital; Surgeon, Eye Dispensary, University Hospital; Instructor in Ophthalmology, Under-Graduate Department, University of Pennsylvania. 1930 Chestnut St.

1905. Carpenter, Herbert B., M.D. 1805 Spruce St.

1926. Carpenter, Howard Childs, M.D., Vice-Dean for Pediatrics and Professor of Pediatric Hygiene, Graduate School of Medicine, University of Pennsylvania; Assistant Professor of Pediatrics, School of Medicine, University of Pennsylvania; Director, Department for the Prevention of Disease, Children's Hospital. 1805 Spruce St.

1895. Carpenter, John T., M.D., Consulting Ophthalmologist to the Bryn Mawr and Abington Hospitals. 1930 Chestnut St.

1917. Carson, John B., M.D., Physician to Episcopal Hospital; Assistant Physician, Pennsylvania Hospital. 1802 Pine St.

1922. Case, Eugene A., M.D., Professor of Morbid Anatomy, Graduate School of Medicine, University of Pennsylvania; Assistant Pathologist, Philadelphia General Hospital. Lansdowne, Pa.

1892. Cattell, Henry W., A.M., M.D., Editor of the International Clinics. Box 311, Burlington, N. J.

1900. Chance, Burton, M.D., Attending Surgeon to the Wills Eye Hospital; Consulting Ophthalmologist Eastern Pennsylvania Institution for the Feeble-minded and Epileptics; Ophthalmic Surgeon to the Pennsylvania Railroad Company. 315 S. Fifteenth St.

1903. Chrystie, Walter, M.D. One of the Senior Physicians to Bryn Mawr Hospital. Bryn Mawr, Pa.

1929. Clark, Eliot R., A.B., M.D., Professor of Anatomy and Director of the Department of Anatomy, School of Medicine, University of Pennsylvania. 301 St. Mark's Square.

1926. Clarke, J. Alexander, A.B., M.D., Chief Clinical Assistant, Out-Patient Department of Applied Immunology, Jefferson Hospital; Assistant Physician, Philadelphia General Hospital; Instructor in Medicine, Undergraduate School, University of Pennsylvania. 334 S. Twenty-first St.

1926. Clerf, Louis H., M.D., Instructor in Bronchoscopy and Esophagoscopy and Demonstrator of Laryngology, Jefferson Medical College; Assistant Professor of Bronchoscopy and Esophagoscopy, Graduate School of Medicine, University of Pennsylvania; Chief Clinical Assistant in Bronchoscopy and Esophagoscopy, Jefferson Hospital; Assistant in Bronchoscopy and Esophagoscopy, University Hospital. 1216 Walnut St.

ELECTED

1910. Cloud, J. Howard, M.D., Attending Physician to the Bryn Mawr Hospital. 11 W. Montgomery Ave., Ardmore, Pa.

1903. Coates, George Morrison, A.B., M.D., Professor of Oto-Rhinology, Graduate School of Medicine, University of Pennsylvania; Surgeon in Charge Department Oto-laryngology, Abington Memorial Hospital; Associate Laryngologist, Pennsylvania Hospital; Consulting Oto-laryngologist to the Philadelphia Orphanage, U. S. Veterans' Bureau and Base Hospital No. 49, Chester County Hospital, Lawrenceville School and to the Sharon Hospital, Sharon, Conn. 1721 Pine St.

1908. Codman, Charles A. E., M.D., Physician to the American Oncologic Hospital. S. E. Cor. Forty-second and Spruce Sts.

1922. Cohen, Leon Solis, A.B., M.D., Chief Clinical Assistant of Roentgenology, Jefferson Medical College; Radiologist to the Memorial Hospital, Roxborough; Radiologist and Director of the Research Tumor Clinic, Jewish Hospital. 1923 Spruce St.

1907. Cohen, Myer Solis, A.B., M.D., Assistant Professor of Medicine, Graduate School of Medicine, University of Pennsylvania; Lecturer on Medical Jurisprudence and Toxicology, Woman's Medical College of Pennsylvania; Pediatrist to the Jewish and Mount Sinai Hospitals. 2110 Spruce St.

1888. Cohen, Solomon Solis, M.D., H.L.D., Emeritus Professor of Clinical Medicine in the Jefferson Medical College; Emeritus Physician to the Jefferson Hospital; Consulting Physician to the Philadelphia General and Jewish Hospitals. 1906 Walnut Street.

1920. Cole, Charles J., A.B., M.D., Visiting Physician, Philadelphia Hospital for Contagious Diseases; Physician to Out-Patient Department, Mary Drexel Home. Elkins Park, Pa.

1898. Coles, Stricker, M.D. Bryn Mawr, Pa.

*1901. Coley, Thomas Luther, A.B., M.D., Major, Medical Corps, U. S. Army. Fort Davis, Canal Zone.

1930. Cooper, David A., B.S., M.D., Instructor in Medicine, University of Pennsylvania. 1520 Spruce St.

†1912. Copp, Owen, A.B., M.D., Consultant for the Future Development of the Pennsylvania Hospital. 4401 Market St.

1911. Cornell, Walter Stewart, A.B., M.D., Director of Medical Inspection of Public Schools of the City of Philadelphia; Chief of Medical Staff of the House of Detention; Lecturer on Osteology in the University of Pennsylvania. Twenty-first and Vine Sts.

1914. Corson, Edward Foulke, M.D., Assistant Professor of Dermatology, Jefferson Medical College; Chief Clinical

ELECTED

Assistant in Dermatology, Jefferson Hospital; Dermatologist, Chestnut Hill Hospital. Medical Arts Building.

1930. Cottrell, James E., M.D., Instructor in Medicine, Medical School, University of Pennsylvania; Assistant Physician, University Hospital; Associate Physician, Episcopal Hospital. 256 S. Twenty-first St.

1924. Cowan, Alfred, M.D., Assistant Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Instructor in Ophthalmology, School of Medicine, University of Pennsylvania; Ophthalmologist, Philadelphia General Hospital. 1930 Chestnut St.

1904. Craig, Frank A., M.D., Instructor in Medicine in the University of Pennsylvania; Visiting Physician to the Henry Phipps Institute, University of Pennsylvania; Visiting Physician to the White Haven Sanatorium; Tuberculosis Consultant to the Presbyterian Hospital. Henry Phipps Institute.

1907. Crampton, George S., M.D., Attending Surgeon to the Eye Department of the Pennsylvania Hospital and the Philadelphia Hospital for Contagious Diseases; Assistant Surgeon to the Wills Hospital; Professor of Ophthalmology in the Graduate School of Medicine, University of Pennsylvania; Ophthalmologist to the Philadelphia Orphanage. 2031 Locust St.

1924. Crawford, Baxter Lindsey, M.D., Associate in Pathology, Jefferson Medical College; Chief of Laboratory Service, Jefferson Hospital; Assistant Pathologist, Philadelphia General Hospital. Jefferson Hospital, Tenth and Sansom Sts.

1920. Creighton, William J., B.A., M.D., Assistant Ophthalmic Surgeon, Germantown Hospital and Dispensary; Assistant Dispensary Surgeon, Department Ophthalmology, University Hospital; Instructor in Ophthalmology, University of Pennsylvania Medical School. 258 S. Eighteenth St.

1920. Cross, George Howard, M.D., Ophthalmologist to the Chester Hospital; Associate in Ophthalmology, Graduate School of Medicine, University of Pennsylvania. Chester, Pa.

1917. Cross, Sumner H., A.B., M.D., Physician to the Abington Memorial Hospital, Abington, Pa. Box 126, Jenkintown, Pa.

1921. Crossan, Edward T., M.D., Associate Surgeon, Episcopal Hospital; Associate in Surgery, University of Pennsylvania; Assistant Surgeon Orthopedic Hospital. 5324 Wayne Ave.

*1924. Crowell, Bowman C., A.B., M.D., C.M., Associate Director, American College of Surgeons; Director of Clinical Research. 40 E. Erie St., Chicago, Ill.

ELECTED

1904. Cruice, John M., M.D., Physician to St. Agnes and St. Joseph's Hospitals and to the Henry Phipps Institute of the University of Pennsylvania; Instructor in Medicine in the University of Pennsylvania. 406 S. Twenty-second St.

*1910. Cummins, W. Taylor, M.D., Pathologist to the Southern Pacific Hospital; Director of the Mary W. Harriman Research Laboratory, San Francisco, Calif.

1902. Currie, Charles A., M.D., Physician to the Germantown Hospital. West Walnut Lane, Germantown.

1896. Da Costa, John Chalmers, M.D., Professor of the Principles of Surgery and of Clinical Surgery in the Jefferson Medical College; Surgeon to the Philadelphia General and St. Joseph's Hospital. 2047 Walnut St.

1887. Daland, Judson, M.D., Professor of Medicine, Graduate School of Medicine, University of Pennsylvania. 317 S. Eighteenth St.

1919. Davies, John R., Jr., M.D., Chief to the Out-Patient Nose, Throat and Ear Department of the Pennsylvania, Bryn Mawr and Chestnut Hill Hospitals. 302 S. Nineteenth St.

1896. Davis, Charles N., M.D., Dermatologist to the Pennsylvania Hospital; Consulting Dermatologist to St. Agnes' Hospital; Assistant Physician to the Dispensary for Skin Diseases in the Howard Hospital. 1931 Spruce St.

1916. Davis, Warren B., M.D., Consulting Oto-laryngologist, St. Agnes' Hospital; Associate in Surgery, Jefferson Medical College; Chief Surgical Assistant in Maxillo-facial Surgery, Division A, Jefferson Hospital; Assistant Surgeon, Frankford Hospital. 135 S. Eighteenth St.

1900. Davisson, Alex. Heron, M.D., Physician to the Rush Hospital; Clinical Chief, City Chest Clinic, No. 12, Rush Hospital.

‡1894. Deaver, Harry C., M.D., Surgeon to the Episcopal Hospital and to the Children's Hospital of the Mary J. Drexel Home; Surgeon-in-Chief to the Kensington Hospital for Women. 337 S. Eighteenth St.

‡1887. Deaver, John B., M.D., D.Sc., LL.D., Professor of Surgery in the Graduate School of Medicine; Emeritus Professor of Surgery in the School of Medicine, University of Pennsylvania; Surgeon-in-Chief to the Lankenau Hospital. 1830 DeLancey Street.

1902. Dehoney, Howard, M.D. 240 S. Thirteenth St.

ELECTED

1924. Deichler, L. Waller, M.D., Assistant Surgeon, Wills Hospital; Instructor in Ophthalmology, University of Pennsylvania; Ophthalmologist, Philadelphia General Hospital. 1930 Chestnut St.

1923. De Long, Perce, M.D., Associate in Ophthalmic Pathology in the Graduate School of Medicine, University of Pennsylvania; Assistant Surgeon, Wills Eye Hospital; Assistant Neuropathologist, Philadelphia General Hospital. 1904 Chestnut Street.

‡1885. Dercum, Francis X., A.M., M.D., Ph.D., Sc.D., President, American Philosophical Society; Professor Emeritus of Nervous and Mental Diseases in the Jefferson Medical College; Foreign Corresponding Member of the Neurological Society of Paris; Honorary Member of the Psychiatric and Neurological Society of Vienna; Corresponding Member of the Neurological Section of the Royal Society of Medicine; Chevalier de l'Ordre National de la Légion d'Honneur. 1719 Walnut St.

1928. Dewees, A. Lovett, B.A., M.D., Attending Physician, Bryn Mawr Hospital. Times Medical Building, Ardmore, Pa.

1912. Dewey, J. Hiland, Ph.B., M.D., Assistant Surgeon to Wills Hospital; Ophthalmic Surgeon to St. Francis' Hospital, Trenton, N. J. 1436 Diamond St.

*1911. Dickson, Frank D., M.D. 1600 Professional Building, Kansas City, Mo.

1908. Dillard, Henry K., Jr., M.D., Physician to the Out-Patient Department of the Pennsylvania Hospital; Physician to the Dispensary of the Mary J. Drexel Home. 234 S. Twentieth Street.

1924. Dillon, Edward S., A.B., M.D., Assistant Physician, Pennsylvania Hospital; Assistant Physician, Department of Metabolism, Philadelphia General Hospital; Instructor in Medicine, School of Medicine, University of Pennsylvania. 2040 Pine St.

1920. Diven, John, A.B., M.D., Pediatrician to St. Christopher's Hospital and Babies' Hospital of Philadelphia. 326 S. Nineteenth St.

1920. Doane, Joseph Chapman, M.D., Associate Professor of Medicine, Temple University; Medical Director, Jewish Hospital; Associate Professor of Medicine, Graduate School of Medicine, University of Pennsylvania. Nineteenth and Spruce Sts.

*1897. Dorland, W. A. Newman, A.M., M.D., Professor of Gynecology in the Post-graduate Medical School of Chicago; Professor of Obstetrics in the Chicago College of Medicine

ELECTED

and Surgery; Visiting Obstetrician to Cook County Hospital. 185 N. Wabash Ave., Chicago, Ill.

1907. Dorrance, George Morris, M.D., Professor of Maxillo-Facial Surgery, Thomas Evans Institute, University of Pennsylvania; Surgeon to St. Agnes' Hospital. 2101 Spruce St.

1902. Downs, Robert N., Jr., M.D., Surgeon to the Dispensary of the Germantown Hospital. 6008 Greene St., Germantown.

1910. Drayton, William, Jr., M.D., Physician to the Philadelphia Hospital for Contagious Diseases; Physician to the Pennsylvania Institute for the Instruction of the Blind. 407 S. Twenty-second Street.

1911. Earnshaw, Henry Culp, M.S., M.D., Attending Physician to the Hospital of the Good Shepherd, Rosemont; Assistant Attending Physician to the Bryn Mawr Hospital; Attending Physician to the Bryn Mawr Children's Hospital; Pennsylvania Railroad Surgeon. Bryn Mawr, Pa.

*1887. Edwards, William A., M.D., Professor of Pediatrics in the Medical Department of the University of California. R. D. 2, Box 105, El Cajon, Calif.

1924. Egbert, Seneca, M.D., Professor of Hygiene, School of Hygiene and Public Health, University of Pennsylvania. Wayne, Pa.

1930. Eiman, John, M.D., Director, Department of Pathology, Presbyterian Hospital; Assistant Professor of Pathology, Graduate School and Associate Department of Pathology, School of Medicine, University of Pennsylvania. Cynwyd, Pa.

1911. Eliason, Eldridge L., B.A., M.D., Sc.D., Professor of Clinical Surgery in the University of Pennsylvania; Surgeon to the University Hospital and to the Howard Hospital; Assistant Surgeon to the Philadelphia General Hospital and the American Stomach Hospital. 326 S. Nineteenth St.

1928. Ellison, Richard T., B.S., M.D., Instructor in Medicine, School of Medicine, University of Pennsylvania; Assistant Physician in Charge of Heliotherapy at the Home for Consumptives, Chestnut Hill; Assistant Physician, Presbyterian Hospital. 111 Rex Ave.

1904. Elmer, Walter G., B.S., M.D., Professor of Orthopedics, Graduate School of Medicine, University of Pennsylvania; Clinical Professor of Orthopedic Surgery in the Woman's Medical College of Pennsylvania; Orthopedic Surgeon to the Hospital of the Graduate School of Medicine and to the Philadelphia General Hospital. 1801 Pine St.

‡1896. Ely, Thomas C., A.M., M.D. 3912 Chestnut St.

ELECTED

†1893. Eshner, Augustus A., M.D., Consulting Physician to Mercy Hospital. 1019 Spruce St.

*1905. Evans, Joseph S., Jr., A.B., M.D., Professor of Medicine, Medical School, University of Wisconsin; Physician to State of Wisconsin General Hospital; Consulting Physician, Madison General and St. Mary's Hospitals. 1300 University Ave., Madison, Wis.

1905. Evans, William, M.D. 500 W. Gay St., West Chester.

1912. Eves, Curtis C., M.D., Chief of Oto-laryngological Service, Pennsylvania Hospital; Oto-laryngologist to Bryn Mawr Hospital; Consultant Oto-laryngologist to the Philadelphia General Hospital. 1910 Spruce St.

1922. Farley, David L., B.S., M.D., Associate in Medicine, University of Pennsylvania; Physician to Pennsylvania Hospital; Chief of Out-Patient Medical Clinic, Pennsylvania Hospital. 1725 Pine St.

†1903. Farr, Clifford B., A.M., M.D., Director of Laboratories, Pennsylvania Hospital, Department for Mental and Nervous Diseases. Bryn Mawr, Pa.

1893. Farr, William W., M.D., Physician to the Leamy Home. 121 Maplewood Ave., Germantown.

1930. Farrell, John T., Jr., B.S., M.D., Assistant Roentgenologist, Jefferson Hospital; Roentgenologist to Daniel Baugh Institute of Anatomy of Jefferson Medical College. 235 S. Fifteenth St.

1907. Fetterolf, George, A.B., M.D., Sc.D., Professor of Oto-laryngology, School of Medicine, University of Pennsylvania. 2010 Spruce St.

1924. Fewell, Alexander G., M.D., Assistant Professor of Ophthalmology, Graduate School of Medicine, and Instructor in Ophthalmology in the School of Medicine, University of Pennsylvania; Ophthalmic Surgeon to the Episcopal Hospital. 1924 Pine St.

1907. Fife, Charles A., A.B., M.D., Associate Professor of Pediatrics in the Graduate School of Medicine of the University of Pennsylvania; Pediatrist to the Presbyterian Hospital; Physician to the St. Christopher's Hospital for Children; Assistant Physician to the Philadelphia General Hospital. 2104 Spruce St.

1884. Fisher, Henry M., A.B., M.D. Jenkintown, Pa.

1910. Fisher, John Monroe, M.D., Associate Professor of Gynecology in the Jefferson Medical College; Gynecologist to the Philadelphia, St. Agnes' and Pottstown Hospitals; Assistant

ELECTED

Gynecologist to the Jefferson Medical College Hospital. 345 S. Nineteenth St.

1928. Fitz-Hugh, Thomas, Jr., A.B., A.M., M.D., Assistant Attending Physician, University Hospital; Attending Physician, Delaware County Hospital; Instructor in Medicine, University of Pennsylvania. 2020 Locust St.

1928. Flick, John Bernard, M.D., Surgeon to Pennsylvania Hospital; Assistant Surgeon to Bryn Mawr Hospital; Associate in Surgery, Jefferson Medical College. 1608 Spruce St.

1888. Flick, Lawrence F., M.D. 736 Pine St.

1916. Forst, John R., M.D. 166 W. Coulter St., Germantown.

1908. Foulkrod, Collin, M.D., Obstetrician to the Presbyterian Hospital; Assistant Demonstrator of Obstetrics in Jefferson Medical College. 3910 Chestnut St.

1908. Fox, Herbert, M.D., Director of the William Pepper Clinical Laboratory and Professor of Comparative Pathology, School of Medicine, University of Pennsylvania; Pathologist to the Laboratory of Comparative Pathology of the Zoölogical Society of Philadelphia; Pathologist to the Rush Hospital. Haverford, Pa.

1906. Fraley, Frederick, Jr., A.B., M.D., Associate in Pediatrics, Graduate School of Medicine, University of Pennsylvania. Overbrook, Pa.

1897. Frazier, Charles H., A.B., M.D., Sc.D., John Rhea Barton Professor of Surgery, University of Pennsylvania. 3400 Spruce St.

1928. Fuller, Daniel Hunt, A.B., A.M., M.D., Medical Director, Department for Men, Pennsylvania Hospital for Mental and Nervous Diseases. 4401 Market St.

1916. Funk, Elmer Hendricks, M.D., Clinical Professor of Medicine and Therapeutics in the Jefferson Medical College; Assistant Physician to the Pennsylvania and Jefferson Hospitals; Visiting Physician to the White Haven Sanatorium. 269 S. Nineteenth St.

1899. Gamble, Robert, G., M.D., one of the Attending Physicians to the Bryn Mawr Hospital. Haverford, Pa.

1931. Garner, Vaughn C., M.D., M.S., Assistant Professor of Dermatology and Syphilology, University of Pennsylvania. Germantown Professional Building.

*1913. Gates, Nathaniel, M.D. 10 Peterboro Ave., Detroit, Mich.

1917. Gerhard, Arthur Howell, M.D. 1615 N. Delaware Ave.

1902. Ghriskey, Albert A., M.D. 3936 Walnut St.

ELECTED

1899. Gibbon, John H., M.D., Emeritus Professor of Surgery in the Jefferson Medical College; Consulting Surgeon to the Pennsylvania and Bryn Mawr Hospitals. 1608 Spruce St.

1920. Gill, A. Bruce, A.B., M.D., Professor of Orthopedics, University of Pennsylvania; Surgeon to the Orthopedic Hospital; Orthopedic Surgeon to the Presbyterian Hospital; Chief Surgeon to the Widener Memorial Industrial Training School for Crippled Children. 1930 Chestnut St.

1931. Gilman, Robert Louis, B.S., M.Sc. (Med.), M.D., Research Assistant, Department of Dermatological Research, University of Pennsylvania; Assistant Professor of Dermatology, Woman's Medical College. 1930 Chestnut St.

1927. Gilmour, William Rodger, A.B., A.M., M.D., Assistant Surgeon, Philadelphia General and Methodist Hospitals; Instructor in Anatomy, University of Pennsylvania; Associate Professor of Operative Surgery and Surgical Anatomy, Woman's Medical College. 1737 Chestnut St.

1931. Giordano, Anthony A. S., M.D., Instructor in Oto-laryngology, School of Medicine, University of Pennsylvania; Instructor in Otology, Graduate School of Medicine, University of Pennsylvania; Assistant in Oto-laryngology, St. Agnes Hospital. 1819 S. Broad St.

1897. Girvin, John H., M.D., Gynecologist to the Presbyterian Hospital; Associate Professor of Gynecology, Graduate School of Medicine, University of Pennsylvania. 2120 Walnut St.

1920. Githens, Thomas Stotesbury, M.D., Chief Pharmacologist, H. K. Mulford Company. The Cambridge, Wissahickon and School Lane, Germantown.

*1906. Gittings, J. Claxton, M.D. Harwood P.O., Md.

1905. Given, Ellis E. W., M.D., Surgeon to the Philadelphia Freemasons Memorial Hospital of the Masonic Home, Elizabethtown, Pa. 2714 Columbia Ave.

1894. Gleason, E. B., S.B., M.D., LL.D., Professor of Otology in the Graduate School of Medicine, University of Pennsylvania. 2112 Pine St.

1906. Goepp, R. Max, M.D. 518 Medical Arts Building.

1906. von Goldberg, Harold G., M.D., Ophthalmic Surgeon to the Episcopal Hospital and Baltimore & Ohio Railroad; Consulting Ophthalmologist, Misericordia and Kensington Hospitals. 2213 Walnut St.

*1908. Goodman, Edward H., M.D. Dorset, Vt.

1905. Gordon, Alfred, M.D., Neurologist to the Mt. Sinai, the Northwestern General, and the Douglass Memorial Hospitals. 1812 Spruce St.

ELECTED

1928. Gordon, Burgess Lee, A.B., M.D., Director of the Department for Diseases of the Chest, Jefferson Hospital; Assistant Professor in Medicine, Jefferson Medical College. 1832 Spruce St.

1894. Graham, Edwin E., M.D., Professor of Pediatrics in the Jefferson Medical College; Pediatrist to the Jefferson and the Philadelphia General Hospitals; Physician to the Franklin Reformatory Home. 1713 Spruce St.

1926. Grant, Francis C., A.B., M.D., Assistant Professor of Neurosurgery and Associate in Surgery, School of Medicine, University of Pennsylvania; Associate in Neuro-Surgery, Graduate School of Medicine, University of Pennsylvania; Visiting Surgeon, Department of Neurological Surgery, Philadelphia General Hospital. 3400 Spruce St.

1904. Grayson, Charles P., M.D., Emeritus Professor of Laryngology in the University of Pennsylvania; Oto-laryngologist to the Philadelphia General Hospital. 262 S. Fifteenth St.

1910. Greenman, Milton J., M.D., Sc.D., Director of the Wistar Institute of Anatomy and Biology. Wistar Institute of Anatomy and Biology, Thirty-sixth St. and Woodland Ave.

1883. Griffith, J. P. Crozer, A.B., M.D., Ph.D., Professor of Pediatrics in the Graduate School of Medicine, University of Pennsylvania; Corresponding Member of the Société de Pédiatrie de Paris; Physician to the Children's Hospital of Philadelphia. 1810 Spruce St.

1912. Griscom, J. Milton, B.S., M.D., Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Attending Surgeon to the Wills Hospital. 2213 Walnut St.

1911. Gummey, Frank Bird, M.D., Visiting Physician to the Germantown Hospital and Dispensary; Visiting Physician to the Midnight Mission. 5418 Greene St., Germantown.

*1902. Gwyn, Norman B., M.D., Clinician in Medicine, Toronto General Hospital; Clinician in Medicine, Medical Faculty, University of Toronto School. 109 Madison Ave., Toronto, Canada.

1894. Hamill, Samuel McC., M.D., Consulting Pediatrist to the Presbyterian Hospital; Pediatrist to St. Vincent's Home. 1822 Spruce St.

1897. Hand, Alfred, Jr., M.D., Professor of Pediatrics, Graduate School of Medicine, University of Pennsylvania; Consulting Physician to the Children's Hospital; Visiting Physician to the Children's Hospital of the Mary J. Drexel Home; Pedi-

ELECTED

atrist to the Widener Memorial School; Consulting Pediatrist to the Methodist Hospital. 1724 Pine St.

1886. Hansell, Howard F., M.D., Emeritus Professor of Ophthalmology in the Jefferson Medical College. N. E. Cor. Seventeenth and Walnut Sts.

‡1889. Hare, Hobart A., M.D., LL.D., Professor of Therapeutics, Materia Medica, and Diagnosis in the Jefferson Medical College. 1801 Spruce St.

1929. Harrison, Francis G., M.D., Assistant Professor of Urology, Graduate School of Medicine, University of Pennsylvania; Assistant Urologist, Out-Patient Department, Presbyterian Hospital. 1900 Spruce St.

1903. Hart, Charles D., A.M., M.D., Inspector and Secretary of the Eastern State Penitentiary; National Executive Committee and Chairman of the Philadelphia Committee, Boy Scouts of America. Chestnut Hill.

1926. Hartman, Frederick L., A.B., M.D., Clinical Associate in Medicine, Lankenau Hospital; Assistant Instructor in Pathology, University of Pennsylvania; Assistant Pathologist, Visiting Staff of University and Philadelphia General Hospitals. 1914 Pine St.

1907. Hatfield, Charles James, A.B. (Princeton), M.D., Executive Director of the Henry Phipps Institute for the Study, Treatment, and Prevention of Tuberculosis; Visiting Physician to the White Haven Sanatorium. Phipps Institute, Seventh and Lombard Sts.

*1928. Hayman, Joseph M., Jr., A.B., M.D., Associate Professor of Medicine, Western Reserve University; Associate Physician, Lakeside Hospital. Cleveland, Ohio.

1911. Heed, Charles R., M.D., Ophthalmologist to Girard College; Assistant Ophthalmologist to the Jefferson Medical College Hospital. 1205 Spruce St.

1901. Heisler, John C., M.D., Professor of Anatomy, University of Pennsylvania. 3829 Walnut St.

1903. Henry, J. Norman, M.D., Physician to the Pennsylvania Hospital; Clinical Professor of Medicine in the Woman's Medical College of Pennsylvania; Assistant Physician to the Philadelphia General Hospital. 1906 Spruce St.

1927. Herman, Leon, B.S., M.D., Urologist, Methodist Hospital; Assistant Surgeon in Charge of Urology, Pennsylvania Hospital. 801 Medical Arts Building.

ELECTED

1891. Hewson, Addinell, A.B., A.M., M.D., Professor of Anatomy and Histology in the Temple University; Surgeon to Memorial Hospital, Roxborough. 257 S. Sixteenth St.

1924. Hewson, William, A.B., M.D., Assistant in Otology, Graduate School of Medicine, University of Pennsylvania; Otolaryngologist, Pennsylvania Institution for Instruction of Blind, Out-Patient Department, Children's Hospital; Assistant Visiting Surgeon Department Oto-laryngology, Abington Memorial Hospital. 6013 Greene St., Germantown.

†1909. Higbee, William S., M.D., President of the Pennsylvania State Board of Examiners for Registration of Nurses. 5121 Pine St.

†1897. Hinkle, William M., M.D., Associate in Otology in the Graduate School of Medicine, University of Pennsylvania; Lecturer on the Anatomy and Physiology of the Vocal Organs in the National School of Elocution and Oratory. 923 N. Sixty-fourth St.

*1892. Hinsdale, Guy, A.M., M.D., Medical Director, White Sulphur Springs, W. Va.

1888. Hirst, Barton Cooke, A.B., M.D., LL.D., Professor of Obstetrics, Graduate School of Medicine, University of Pennsylvania; Gynecologist to the Philadelphia General Hospital. 1821 Spruce St.

1924. Hirst, John C., 2nd, A.B., M.D., Assistant Professor of Obstetrics, School of Medicine and Graduate School of Medicine, University of Pennsylvania; Assistant Obstetrician, University Hospital; Obstetrician to St. Agnes' Hospital; Gynecologist to Orthopedic Hospital. 1918 Pine St.

*1908. Hitchens, Arthur Parker, M.D., M.C., U.S.A., in charge, 6th Corps Area. Laboratory, Fort Sheridan, Ill.

1905. Hodge, Edward Blanchard, A.B., M.D., Surgeon to the Presbyterian Hospital; Chief of Surgical Service, Germantown Hospital; Surgeon-in-Chief to Chester County Hospital, West Chester, Pa. 2019 Spruce St.

1931. Holland, H. Albert, M.D., Assistant Chief, Medical Staff, Chestnut Hill Hospital. 8200 Crittenden St., Chestnut Hill.

1906. Holloway, Thomas B., M.S., M.D., Sc.D., Professor of Ophthalmology, School of Medicine, University of Pennsylvania; Ophthalmologist, Orthopedic Hospital, and in Department of Neurology, Philadelphia General Hospital; Ophthalmologist to the Pennsylvania Institution for the Instruction of the Blind at Overbrook. 1819 Chestnut St.

ELECTED

‡1914. Hooker, Richard S., M.D., Assistant Physician to the Episcopal Hospital, and to the Philadelphia Orthopedic Hospital and Infirmary for Nervous Diseases. 2147 N. Howard St.

1919. Hopkins, Arthur H., B.S. (Haverford), M.D. 1726 Pine St.

1925. Hopkinson, R. Dale, M.D., Registrar and Assistant Physician, Abington Memorial Hospital. Jenkintown, Pa.

1928. Houser, Karl M., Ph.B., M.D., M.Sc., Instructor in Otolaryngology, University of Pennsylvania; Assistant in Otolaryngology, University Hospital; Oto-laryngologist, Delaware County Hospital. 1826 Pine St.

1908. Hoyt, Daniel M., M.D., Assistant Visiting Physician to the Philadelphia General Hospital. The Tracy, Thirty-sixth and Chestnut Sts.

*1912. Huber, G. Carl, M.D., Professor of Anatomy and Director of the Anatomic Laboratories and Dean of the Graduate School, University of Michigan. 1330 Hill St., Ann Arbor, Mich.

1892. Hughes, William E., M.D., Visiting Physician to the Philadelphia General Hospital; Consulting Physician to the Presbyterian Hospital. 3945 Chestnut St.

1923. Hume, John E., A.B., M.D. N. E. Cor. Twentieth and Chestnut Sts.

*1912. Hunter, John W., B.S., M.D. McDaniel, Md.

1921. Hunter, Robert J., M.D., Laryngologist, Philadelphia General Hospital; Associate Professor of Laryngology, Graduate School of Medicine, University of Pennsylvania. 2011 Chestnut Street.

1926. Husik, David N., M.D., Professor of Otology, Graduate School of Medicine, University of Pennsylvania; Laryngologist, Philadelphia General, Mount Sinai and American Stomach Hospitals. 1930 Chestnut St.

1898. Hutchinson, James P., M.D., Consulting Surgeon to the Pennsylvania and to the Methodist Hospitals. Media, Pa.

1921. Ivy, Robert H., D.D.S., M.D., Professor of Maxillo-Facial Surgery, Graduate School of Medicine, University of Pennsylvania; Plastic Surgeon to the Hospitals of the Graduate School of Medicine, University of Pennsylvania; Oral Surgeon, Episcopal Hospital; Consultant in Maxillo-Facial Surgery, Walter Reed General Hospital, Washington, D.C. 1930 Chestnut St.

ELECTED

1917. Jackson, Chevalier, M.D., Sc.D., LL.D., Professor of Bronchoscopy and Esophagoscopy, Temple University School of Medicine; Bronchoscopist and Esophagoscopist, Temple University Hospital; William Potter Memorial Lecturer, Jefferson Medical College; Lecturer on Bronchoscopy and Esophagoscopy, Woman's Medical College. 3432 N. Broad St.

*1885. Jackson, Edward, A.M., M.D., Sc.D., Emeritus Professor of Ophthalmology in the University of Colorado; Emeritus Professor of Diseases of the Eye in the Philadelphia Polyclinic; Secretary of State Commission for the Blind of Colorado. 217 Imperial Building, Denver, Colo.

1906. Jacobs, Francis Brinton, B.S., M.D., Pediatrist to the Chester County Hospital. West Chester, Pa.

1922. Jameson, Howard Leon, M.D., Associate Professor of Medicine, Graduate School of Medicine, University of Pennsylvania. 2133 S. Sixteenth St.

‡1920. Janvier, G. Victor, M.D., Associate in Obstetrics, School of Medicine, University of Pennsylvania; Assistant Obstetrician, University Hospital; Assistant Obstetrician and Gynecologist, Philadelphia General Hospital; Cystoscopist to the Obstetric and Gynecologic Department of the Delaware County Hospital. Lansdowne, Pa.

1913. Jefferys, William Hamilton, A.B., A.M., M.D. Rosemont, Pa.

‡1919. Jenks, Horace H., A.B., M.D., Medical Director, Associated Medical Clinics; Assistant Visiting Physician to the Children's Hospital; Assistant Professor of Pediatrics, Graduate School of Medicine, University of Pennsylvania; Physician to St. Christopher's Hospital for Children. 918 Clinton St.

1930. Jepson, Paul Newton, A.B., M.D., M.S. 1824 Spruce St.

1928. John, Rutherford Lewis, B.S., M.D., Instructor in Surgery, School of Medicine, University of Pennsylvania; Associate Surgeon in Charge of Orthopedics, Episcopal Hospital; Orthopedic Surgeon to St. Christopher's Hospital; Assistant Surgeon to the Orthopedic Hospital and Infirmary for Nervous Diseases. 256 S. Twenty-first St.

1926. Johnson, George Ernest, M.D. 5341 Chester Ave.

1898. Johnson, Russell H., A.B. (Princeton), M.D., Physician to the Pennsylvania Institution for the Deaf and Dumb. Chestnut Hill, Philadelphia.

†1928. Johnson, William N., A.B., M.D., Consulting Physician, Germantown Hospital. 6430 Germantown Ave.

ELECTED

1918. Jonas, Leon, M.D. 1726 Pine St.

†1900. Jones, Charles James, A.M., M.D., LL.D., Ophthalmic Surgeon to St. Joseph's Hospital; Ophthalmic Surgeon to the House of the Good Shepherd, Germantown; Consulting Ophthalmologist to St. Vincent's Home. 626 Medical Arts Building.

1924. Jones, Harold W., M.D., Demonstrator of Medicine, Jefferson Medical College; Assistant Physician, Philadelphia General Hospital. 1930 Chestnut St.

1913. Jones, John F. X., B.S., A.B., A.M., M.D., Surgeon to St. Joseph's, Misericordia and St. Agnes' Hospitals. 1737 Chestnut St.

1900. Jopson, John H., M.D., Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Surgeon to the Presbyterian Hospital; Consulting Surgeon to Children's and Bryn Mawr Hospitals. 1824 Pine St.

1900. Judson, Charles F., A.B., M.D., Physician to Out-Patient Department of the Lankenau Hospital and to the Southern Home for Children. 1005 Spruce St.

1902. Jump, Henry D., M.D., Professor of Applied Therapeutics and Clinical Medicine, Woman's Medical College; Physician to Philadelphia General and Misericordia Hospitals. 2019 Walnut St.

1903. Kalteyer, Frederick J., M.D., Associate Professor of Medicine, Jefferson Medical College; Assistant Physician, Jefferson Medical College Hospital; Consulting Physician to the Pottstown Hospital, Pottstown, Pa. 1530 Locust St.

*1910. Karsner, Howard T., M.D., Professor of Pathology and Director of the Institute of Pathology, Western Reserve University. 2109 Adelbert Rd., Cleveland, Ohio.

1926. Kaufman, Isadore, M.D., Associate in Medicine, University of Pennsylvania; Medical Director, Kensington Dispensary for Tuberculosis; Associate Clinical and Sociological Director, Phipps Institute; Visiting Physician to White Haven Sanatorium. 1616 Walnut St.

1923. Kay, James, M.D., Visiting Physician, Episcopal Hospital and to the Philadelphia Hospital for Mental Diseases, Byberry; Associate Professor in Physical Diagnosis, Medical School, Temple University; Consulting Physician to Kensington Hospital for Women. 600 W. Olney Ave.

1924. Keeler, J. Clarence, M.D., Associate Professor of Otology, Jefferson Medical College and Hospital; Assistant in Oto-

ELECTED

laryngology, Germantown Hospital; Consultant Otolaryngologist, Pottstown Hospital, Pa., and Newcombe Hospital, Vineland, N. J. Medical Arts Building.

†1867. KEEN, WILLIAM W., M.D., LL.D. (Hon.), Ph.D. (Brown, Upsala, etc.), (Hon.) Sc.D. (Jefferson and Harvard), Dr., Honoris Causa, University of Paris; (Hon.) F.R.C.S. (Eng., Edin. and Ireland). 1520 Spruce St.

1912. KEENE, FLOYD E., M.D., Goodell Professor of Gynecology in the School of Medicine, and Professor of Gynecology in the Graduate School of Medicine, University of Pennsylvania; Gynecologist to the University Hospital; Gynecologist to the Chestnut Hill Hospital. 133 S. Thirty-sixth St.

1913. KELLY, FRANCIS JOSEPH, M.D. 2211 Locust St.

*1887. KELLY, HOWARD A., A.B., M.D., LL.D. (Aberdeen, Wash. and Lee, and Univ. of Pa.), Professor of Gynecology in Johns Hopkins University and Gynecologist to the Johns Hopkins Hospital, Baltimore, Md.; Hon. Fellow of the Edinburgh Obstetrical Society, the Royal Academy of Medicine of Ireland, and of the Glasgow Obstetrical and Gynecological Society. 1418 Eutaw Place, Baltimore, Md.

1909. KELLY, JAMES A., A.M., M.D., Associate Professor of Surgery in the Graduate School of Medicine, University of Pennsylvania; Visiting Surgeon to St. Mary's, St. Joseph's and Misericordia Hospitals. 1815 Spruce St.

1912. KELLY, THOMAS C., A.M., M.D., Associate in Medicine, University of Pennsylvania; Assistant Professor of Medicine, Graduate School, University of Pennsylvania; Pediatrist to St. Christopher, Misericordia and St. Mary's Hospitals; Assistant Physician, Philadelphia General Hospital. 105 School Lane, Germantown.

1898. KEMPTON, AUGUSTUS F., M.D. 2118 Pine St.

1905. KERCHER, DELNO E., M.D. 1534 Pine St.

1921. KERN, RICHARD A., A.B., M.D., Assistant Professor of Medicine, University of Pennsylvania; Assistant Physician, University Hospital; Associate in Medicine, Graduate School of Medicine, University of Pennsylvania. University Hospital, Thirty-sixth and Spruce Sts.

1930. KIMBROUGH, ROBERT A., JR., A.B., M.D., Associate in Obstetrics and Gynecology, University of Pennsylvania; Assistant Gynecologist, University Hospital; Obstetrician, Pennsylvania Hospital. 1907 Spruce St.

1931. KINNEY, WILLARD H., M.D., Genito-urinary Surgeon, Philadelphia General Hospital; Assistant Attending Genito-urinary

ELECTED

Surgeon, Jefferson Hospital; Associate Professor Genito-urinary Surgery, Jefferson Medical College. 315 S. Seventeenth St.

1920. Klauder, Joseph Victor, A.B., M.D., Professor of Dermatology, Woman's Medical College; Associate Professor, Dermatology and Syphilology, Graduate School of Medicine, University of Pennsylvania; Dermatologist, Misericordia, Wills and Abington Memorial Hospitals; Chief of Clinic, Philadelphia General Hospital. 1934 Spruce St.

1920. Klein, Thomas, M.D., Professor of Applied Therapeutics, Medical School, Temple University; Associate Professor of Medicine, Graduate School, University of Pennsylvania; Physician to Samaritan and Philadelphia General Hospitals. 250 S. Eighteenth St.

1913. Klopp, Edward J., M.D., Professor of Surgery in the Jefferson Medical College; Attending Surgeon to the Jefferson Hospital; Surgeon and Chief of the Out-Patient Clinic of the Pennsylvania Hospital; Surgeon to the Delaware County and Memorial Hospitals. 1611 Spruce St.

1908. Knipe, Jay C., M.D., Ophthalmologist to the Jewish and Mary J. Drexel Hospitals; Associate in Ophthalmology, Jefferson Medical College; Chief of Eye Clinic, Jefferson Hospital. 1737 Chestnut St.

1919. Knipe, Norman Leslie, Ph.B. (Cornell); M.D., Gynecologist, St. Mary's Hospital; Associate Gynecologist, American Stomach Hospital. 1930 Chestnut St.

1908. Knowles, Frank Crozer, M.D., Professor of Dermatology, Jefferson Medical College; Dermatologist to the Presbyterian and Children's Hospitals; Chief of Clinic, Pennsylvania Hospital. 2035 Spruce St.

1925. Knox, Harry E., M.D., Surgeon to St. Christopher's Hospital; Surgeon to Philadelphia Hospital for Contagious Diseases; Associate Surgeon, Episcopal Hospital; Assistant Surgeon, Germantown Hospital. 719 Sixty-sixth Ave., Oak Lane, Phila.

1914. Kolmer, John A., M.D., Dr. P.H., M.Sc., D.Sc., LL.D., Professor of Pathology and Bacteriology, Graduate School of Medicine, University of Pennsylvania; Head of the Department of Pathology and Bacteriology Research Institute for Cutaneous Medicine; Consulting Pathologist to St. Vincent's, Misericordia, Memorial and Jeanes Hospitals. Cynwyd, Pa.

1904. Krauss, Frederick, M.D., Ophthalmic Surgeon to the Episcopal Hospital; Ophthalmic Surgeon to St. Christopher's

ELECTED

Hospital for Children; Laryngologist to the Abington Hospital. 1930 Chestnut St.

1914. Krumbhaar, Edward B., A.B., Ph.D., M.D., Professor of Pathology, School of Medicine, and Graduate School of Medicine, University of Pennsylvania. Box 4310, Chestnut Hill.

1900. Krusen, Wilmer, M.D., Professor of Gynecology in the Medical Department of Temple University; Chief Gynecologist to the Samaritan and the Garretson Hospitals; Consulting Gynecologist to the Charity and Mercy Hospitals. Lansdowne, Pa.

1904. Landis, Henry R. M., M.D., Professor of Clinical Medicine in the School of Medicine, and Director of the Clinical and Sociological Departments of the Henry Phipps Institute of the University of Pennsylvania; Visiting Physician to the White Haven Sanatorium. Phipps Institute, Seventh and Lombard Sts.

1907. Langdon, H. Maxwell, M.D., Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Instructor of Ophthalmology, School of Medicine, University of Pennsylvania; Ophthalmologist of the Presbyterian, Orthopedic and Children's Hospitals. 1530 Locust St.

1922. Lawrance, Stuart J., M.D., Obstetrician, St. Mary's Hospital. 405 S. Forty-second St.

1926. Laws, George M., B.S., M.D., Associate Gynecologist, Presbyterian Hospital; Gynecologist, American Hospital for Diseases of the Stomach. 1907 Spruce St.

1920. Leavitt, Frederick Headley, M.D., Associate in Neurology, Graduate School of Medicine, University of Pennsylvania; Assistant Physician to the Orthopedic Hospital and Infirmary for Nervous Diseases. 1527 Pine St.

1904. Le Boutillier, Theodore, M.D., Assistant Professor, Epidemic Contagious Diseases, Graduate School of Medicine, University of Pennsylvania; Senior Pediatrist, Philadelphia Hospital for Contagious Diseases. 1930 Chestnut St.

1908. Lee, Walter Estell, M.D., Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Surgeon to the Pennsylvania, Germantown, Bryn Mawr, Children's and Burlington County Hospitals, and to the Hospital of the Graduate School of Medicine, University of Pennsylvania. 905 Pine St.

1892. Leidy, Joseph, M.D., Officier d'instruction publique, France; Consulting Physician to the Pennsylvania Training School for Feeble-minded Children. 1713 Walnut St.

ELECTED

1920. Leopold, Simon S., M.D., Associate in Medicine, University of Pennsylvania; Assistant Physician, University Hospital. 2025 Spruce St.

1915. Lewis, Fielding O., M.D., Associate in Laryngology in the Jefferson Medical College Hospital; Operating Clinical Chief of the Laryngological Dispensary and Clinical Assistant of the Otological Dispensary of the Jefferson Medical College Hospital; Laryngologist of the Philadelphia General Hospital. 259 S. Seventeenth St.

1904. Lindauer, Eugene, M.D., Assistant Neurologist to the Philadelphia General and Polyclinic Hospitals; Instructor in Neurology, Graduate School of Medicine, University of Pennsylvania. 6451 N. Broad St.

1927. Lindsey, Walter H., A.B., M.D., Associate Physician, Episcopal Hospital; Assistant Medical Director, Philadelphia Rapid Transit Company. S. E. Cor. Seventeenth and Arch Streets.

1886. Lloyd, J. Hendrie, A.M. (Princeton), M.D., Consulting Neurologist to the Methodist Episcopal Hospital; Consulting Neurologist to the State Asylum for the Chronic Insane at Wernersville, and to the Pennsylvania Training School for Feeble-minded Children at Elwyn. 4057 Spruce St.

1907. Lodholz, Edward, M.D., Isaac Ott Professor of Physiology, Graduate School of Medicine, University of Pennsylvania. Medical Laboratories, University of Pennsylvania.

1931. Loewenberg, Samuel A., M.D., Associate Professor of Medicine, Jefferson Medical College; Assistant Visiting Physician Jefferson Hospital; Visiting Physician, Philadelphia General and Northern Liberties Hospitals and to Eaglesville Sanitorium. 1905 Spruce St.

1893. Longaker, Daniel, M.D., Consulting Obstetrician to the Kensington Hospital for Women. 1806 Spruce St.

1931. Longaker, Edwin P., M.D., Instructor in Oto-laryngology, Graduate School of Medicine, University of Pennsylvania; Assistant Laryngologist, Bryn Mawr Hospital; Assistant Laryngologist and Otologist, Out-Patient Department, Pennsylvania Hospital. 1806 Spruce St.

1921. Lucké, Baldwin, Dr. P.H., M.D., Assistant Pathologist, University and Philadelphia General Hospitals; Associate Professor of Pathology, School of Medicine, and Assistant Professor of Pathology in the Graduate School of Medicine,

ELECTED

University of Pennsylvania. Medical School, University of Pennsylvania.

1924. Ludy, John B., A.B., A.M., M.D., Dermatologist to Pennsylvania, Lankenau, Episcopal, Methodist, Philadelphia General, Delaware County and Oncologic Hospitals. Medical Arts Building.

1923. Lueders, Charles W., A.B., M.D., Gastroenterologist and Chief of Clinic, Pennsylvania Hospital. 1930 Chestnut St.

1930. Lukens, Robert M., M.D. 1308 Hunting Park Ave.

1929. Lull, Clifford B., M.D., Demonstrator in Obstetrics, Jefferson Medical College; Attending Physician, Department of Gynecology and Obstetrics, Philadelphia General and Delaware County Hospitals; Assistant Obstetrician, Lying-in and Jefferson Hospitals. 1731 Pine St.

1919. Lynch, Frank B., Jr., M.D., Dickson Fellow in Bacteriology to the Pepper Laboratory of Clinical Medicine, University of Pennsylvania; Serologist, University Hospital; Instructor in Pathology, Evans Institute, University of Pennsylvania. Pepper Laboratory, University of Pennsylvania.

1914. Lyon, B. B. Vincent, A.B. and D.Sc. (Williams Coll.), M.D. (Johns Hopkins), Associate in Medicine, Jefferson Medical College; Chief-of-Clinic, Gastro-Intestinal Department, Jefferson Hospital. 2031 Locust St.

1900. McCarthy, Daniel J., M.D., Professor of Medical Jurisprudence (George B. Wood Foundation) in the University of Pennsylvania; Neurologist to the Philadelphia General and St. Agnes' Hospitals, and to the Henry Phipps Institute. 2025 Walnut St.

1931. McCloskey, Edward, M.D. 7 E. Chestnut Ave.

1913. McCrae, Thomas, B.A., M.D., F.R.C.P. (Lond.), Professor of Medicine in the Jefferson Medical College; Physician to the Jefferson and Pennsylvania Hospitals. 1929 Spruce Street.

1895. McFarland, Joseph, M.D., Sc.D., Professor of Pathology in the School of Medicine, University of Pennsylvania; Pathologist to the Philadelphia General Hospital. 542 W. Hortter St., Germantown.

1931. McIver, Joseph, M.D., Associate Neurologist, University of Pennsylvania; Neurologist, Misericordia Hospital; Visiting Psychiatrist, Philadelphia General Hospital. 133 S. Thirty-sixth St.

ELECTED

1905. McKenzie, R. Tait, M.D., LL.D., Emeritus Professor of Physical Education and Director of the Department of Physical Education in the University of Pennsylvania. 2014 Pine Street.

*1916. McKnight, Howard A., A.B., M.D. Tecumseh Building, Springfield, Ohio.

†1915. McLean, John D., M.D. 135 S. Thirty-sixth St.

1924. McMillan, Thomas M., A.B., M.D., Assistant Physician, Pennsylvania Hospital; Cardiologist, Philadelphia General Hospital; Instructor in Medicine, School of Medicine, University of Pennsylvania; Associate Professor of Cardiology, Graduate School of Medicine, University of Pennsylvania. 2044 Locust St.

1924. McPhedran, F. Maurice, A.B., M.D., Associate in Medicine, School of Medicine, University of Pennsylvania; Assistant Professor of Medicine, Henry Phipps Institute. Seventh and Lombard Sts.

*1900. McReynolds, Robert Phillips, M.D. 213 S. Broadway, Los Angeles, Calif.

1910. Mackinney, William H., M.D., Professor of Urology, Graduate School of Medicine, University of Pennsylvania; Genitourinary Surgeon, Philadelphia General Hospital; Chief of Urologic Clinic, Lankenau Hospital. 1930 Chestnut St.

‡1901. MacLeod, George I., M.D. 131 S. Twenty-second St.

1914. Maier, F. Hurst, M.D., Associate in Gynecology to the Jefferson Medical College; Gynecologist to St. Joseph's Hospital; Gynecologist, Obstetrician and Consultant, Prenatal Clinic, Philadelphia General Hospital; Associate Gynecologist, St. Mary's Hospital. 2019 Walnut St.

1913. Manges, Willis F., D.Sc., M.D., Roentgenologist to the Jefferson Hospital; Clinical Professor of Roentgenology, Jefferson Medical College. 235 S. Fifteenth St.

1898. Marshall George Morley, M.D., Laryngologist to the Philadelphia General Hospital; Laryngologist and Otologist to St. Joseph's Hospital; Associate Professor of Laryngology in the Graduate School of Medicine, University of Pennsylvania. 1819 Spruce St.

1889. Martin, Edward M.D., Emeritus Professor of Surgical Physiology, University of Pennsylvania. 135 S. Eighteenth St.

*1911. Meigs, Edward Browning, A.B., M.D., Physiologist in the Dairy Division of the United States Department of Agriculture. 1736 M. St., N. W., Washington, D. C.

ELECTED

1914. Mencke, J. Bernhard, A.B., M.D., Surgeon, Northwestern General Hospital; Associate Surgeon to the Lankenau Hospital. 1816 Spruce St.

1923. Mendelson, Walter, M.D. 639 Church Lane, Germantown.

1914. Merrill, William Jackson, A.B., M.D., Associate Professor of Orthopedics, Graduate School of Medicine, University of Pennsylvania; Orthopedic Surgeon to the Children's Hospital, Jewish Hospital, Misericordia Hospital; St. Edmond's Home for Crippled Children; Children's Seashore House, Lock Haven Hospital, City Hospital, Williamsport, Pa., and Geisinger Memorial Hospital, Danville, Pa. 2017 Spruce St.

*1894. Miller, D. J. Milton, M.D., Associate Physician to the Children's Hospital, Philadelphia; Pediatrist to the Bamberger Home for Invalid Children, Longport, N. J. N. W. Cor. Pacific and California Aves., Atlantic City, N. J.

1922. Miller, Edwin B., M.D., Ophthalmic Surgeon, Stetson Hospital. 269 S. Nineteenth St.

1921. Miller, T. Grier, A.B., M.D., Assistant Professor of Medicine, University of Pennsylvania; Chief, Gastro-intestinal Clinic; Assistant Physician, University Hospital; Consultant, Friends' Hospital and Shriners' Hospitals for Crippled Children. 318 University Hospital.

‡1881. Mills, Charles K., M.D., LL.D., Emeritus Professor of Neurology in University of Pennsylvania; Neurologist to Philadelphia General Hospital; Consulting Physician to the Orthopedic Hospital and Infirmary for Nervous Diseases. 2121 DeLancey St.

*1917. Mitchell, A. Graeme, M.D., Professor of Pediatrics, College of Medicine, University of Cincinnati; Director of Pediatric and Contagious Services, Cincinnati General Hospital; Chief of Staff of Children's Hospital, Cincinnati. University of Cincinnati.

1904. Mitchell, Charles F., M.D., Chief of Service "A," Pennsylvania, Bryn Mawr and Germantown Hospitals; Consulting Surgeon, Chestnut Hill and St. Christopher's Hospitals. 2003 Pine St.

1921. Mohler, Henry K., P.D., M.D., Associate in Medicine, Jefferson Medical College; Medical Director, Jefferson Hospital. 319 S. Sixteenth St.

*1908. Montgomery, Charles M., A.B., M.D. Oteen, N. C.

1928. Moon, Virgil Holland, A.B., M.D., M.Sc. Jefferson Medical College.

1923. Moore, William F., M.D., Assistant, Bronchoscopic Clinic, Jefferson Hospital; Bronchoscopist to Philadelphia General

ELECTED

Hospital and Philadelphia Hospital for Contagious Diseases; Assistant Laryngologist, Children's Hospital, Out-Patient Department; Laryngologist to Tuberculosis Department, Philadelphia General Hospital. 255 S. Sixteenth St.

1924. Moorhead, Stirling W., A.B., M.D., Associate in Urology, School of Medicine, University of Pennsylvania; Assistant Surgeon, Department of Genito-urinary Surgery and in Urological Dispensary, University Hospital. 1523 Pine St.

1918. Morgan, Arthur C., M.D., Emeritus Professor of Clinical Medicine, Temple University; Visiting Physician to Tuberculosis Department, Philadelphia General Hospital; Consulting Physician to the Eastern State Penitentiary and to the Frankford Hospital; Member Pennsylvania State Board of Medical Education and Licensure. 1930 Chestnut St.

1886. Morris, Caspar, M.D. Haverford, Pa.

1893. Morris, Elliston J., M.D., Consulting Physician to the Episcopal Hospital. 128 S. Eighteenth St.

1929. Mudd, Stuart, B.S., M.A., M.D., Associate Professor of Bacteriology, School of Medicine, University of Pennsylvania. Haverford, Pa.

1905. Muller, George P., M.D., Professor of Clinical Surgery, University of Pennsylvania; Surgeon to the University and Misericordia Hospitals and Mary J. Drexel Home. 1930 Spruce St.

1930. Murphy, Douglas P., M.D., Fellow in Research, Gynecean Hospital Institute of Gynecological Research, University of Pennsylvania. 4422 Osage Ave.

*1915. Musser, John H., Jr., B.S., M.D., Professor of Medicine and Head of Department, School of Medicine, Tulane University of Louisiana. 1427 Second St., New Orleans, La.

1905. Mutschler, Louis H., M.D., Surgeon to the Episcopal Hospital; Associate Surgeon to the Orthopædic Hospital. 1625 Spruce Street.

1902. Nassau, Charles F., M.D., LL.D., Clinical Professor of Surgery in the Jefferson Medical College; Surgeon to St. Joseph's Hospital; Chief Surgeon to the Frankford Hospital; Surgeon to Girard College. 1710 Locust St.

1887. Neilson, Thomas Rundle, A.M., M.D., Surgeon Emeritus to the Episcopal Hospital; Consulting Surgeon to St. Christopher's Hospital for Children; Emeritus Professor of Genito-urinary Surgery in the University of Pennsylvania. 1937 Chestnut St.

ELECTED

1905. Newcomet, William S., M.D., Physician in Charge of Radiological Department, Jefferson, Presbyterian and Oncologic Hospitals. 3501 Baring St.

1905. Newlin, Arthur, B.S., M.D., Physician to the Pennsylvania Hospital. 1804 Pine St.

1899. Nicholson, William Rufus, A.B., M.D., Professor of Gynecology, Graduate School of Medicine, University of Pennsylvania; Attending Gynecologist and Attending Obstetrician to the Methodist Episcopal Hospital; Attending Obstetrician to the Presbyterian Hospital. 2023 Spruce St.

1889. Noble, Charles P., M.D., Sc.D. (Hon.), Formerly Surgeon-in-Chief, Kensington Hospital for Women; Formerly Clinical Professor of Gynecology, Woman's Medical College. Radnor, Pa.

1905. Norris, Charles C., M.D., Director of the Department and Professor of Obstetrics and Gynecology in the School of Medicine, and Professor of Gynecology in the Graduate School of Medicine, University of Pennsylvania; Gynecologist and Obstetrician to the University Hospital; Director, Gynecean Hospital Institute of Gynecological Research, University of Pennsylvania; Gynecologist to Henry Phipps Institute and to the Children's Hospital. 133 S. Thirty-sixth St.

*1905. Norris, George William, A.B., M.D., U. S. Public Health Service (Veterans' Bureau). Dimock, Pa.

*1901. Norris, Henry, M.D., Surgeon to the Rutherford Hospital. Waverly Mills, S. C.

1892. Norris, Richard C., M.D., Professor of Obstetrics in the Graduate School of Medicine, University of Pennsylvania; Obstetrician in Charge of the Preston Retreat; Visiting Obstetrician to the Philadelphia General Hospital; Gynecologist to the Methodist Episcopal Hospital, and Consulting Obstetrician and Attending Gynecologist to the Southeastern Dispensary and Hospital. 500 N. Twentieth St.

1913. O'Neal, Alexander H., A.B., A.M., M.D., Physician to the Catheart and Richardson Homes, Devon; Anesthetizer to the Bryn Mawr Hospital. St. Davids, Pa.

1924. Opie, Eugene L., A.B., M.D., Professor of Pathology and Director of the Department of Pathology in the School of Medicine, University of Pennsylvania; Director of Laboratories, Henry Phipps Institute. Phipps Institute, Seventh and Lombard Sts.

1913. Outerbridge, George W., A.B., M.D., Associate Professor of Gynecology, Graduate School of Medicine, University

ELECTED

of Pennsylvania; Assistant Gynecologist to the Methodist Hospital; Gynecologist to the Abington Memorial Hospital. 1927 Spruce St.

1915. Owen, Hubley R., M.D., Professor of Clinical Surgery, Woman's Medical College; Surgeon, Philadelphia General Hospital; Assistant Surgeon, Orthopedic Hospital; Chief Surgeon, Bureaus of Police and Fire, Philadelphia. 319 S. Sixteenth St.

1897. Packard, Francis R., M.D., Consulting Laryngologist, Pennsylvania Hospital; Consulting Oto-laryngologist to the Children's and Bryn Mawr Hospitals, Editor, *Annals of Medical History*. 304 S. Nineteenth St.

1898. Page, Henry F., M.D., Chief Medical Service "A," Lankenau Hospital; Associate Professor of Medicine, Graduate School of Medicine, University of Pennsylvania. 315 S. Sixteenth St.

1906. Pancoast, Henry K., M.D., Professor of Roentgenology in the School of Medicine, University of Pennsylvania and Roentgenologist to the University Hospital. Haverford, Pa.

1909. Parish, Benjamin D., B.S., M.D., Associate Professor in Otology, Graduate School of Medicine, University of Pennsylvania; Aurist and Laryngologist to St. Agnes' and Chestnut Hill Hospitals. 1927 Spruce St.

1899. Parke, William E., M.D., Consulting Obstetrician to the Episcopal Hospital; Associate Surgeon to the Kensington Hospital for Women; Gynecologist to the Frankford Hospital. 1534 N. Fifteenth St.

1925. Patten, Clarence A., M.D., Assistant Professor of Neurology, Graduate School of Medicine, University of Pennsylvania; Visiting Neurologist, Philadelphia General Hospital; Neurologist, Lankenau Hospital and Delaware General Hospital. 2037 DeLancey St.

1910. Patterson, Ross Vernet, M.D., Dean and Associate Professor of Medicine, Jefferson Medical College; Physician to the Philadelphia General Hospital; Physician to the Protestant Episcopal Hospital; Assistant Physician to the Jefferson Hospital. 2126 Spruce St.

1931. Paul, John Davis, A.B., M.D., Associate Visiting Physician and Associate Physician in charge of Diabetics, Episcopal Hospital; Pathologist to St. Christopher's Hospital. 3112 N. Broad St.

*1926. Paul, John Rodman, A.B., M.D., Assistant Professor of Medicine, School of Medicine, Yale University; Associate

ELECTED

Physician, New Haven Hospital and Dispensary. Yale University, New Haven, Conn.

1922. Pelouze, P. S., M.D., Associate in Urology, School of Medicine, University of Pennsylvania; Surgeon-in-Charge, Urological Dispensary, University Hospital. 1737 Chestnut St.

1909. Pemberton, Ralph, M.S., M.D., Professor of Medicine, Graduate School, University of Pennsylvania; Visiting Physician and Director of the Department of Metabolic Diseases, Presbyterian Hospital; Physician to Orthopedic Hospital. Paoli, Pa.

1930. Pendergrass, Eugene P., M.D., Assistant Professor of Roentgenology, School of Medicine and Assistant Professor of Radiology, Graduate School of Medicine, University of Pennsylvania; Assistant Roentgenologist, University Hospital. Ardmore, Pa.

1914. Pepper, O. H. Perry, B.S., M.D., Professor of Clinical Medicine, School of Medicine, University of Pennsylvania. University Hospital, Thirty-sixth and Spruce Sts.

†1902. Pepper, William, A.B., M.D., Dean of the School of Medicine, University of Pennsylvania; Member of the Council on Medical Education and Hospitals of the American Medical Association. University of Pennsylvania.

1916. Percival, Milton Fraser, M.D. 2332 S. Broad St.

1917. Peter, Luther C., A.M., M.D., Professor of Diseases of the Eye, Temple Medical School; Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania. 1930 Chestnut St.

1912. Petty, Orlando H., B.S., A.M., M.D., Professor of Diseases of Metabolism, Graduate School of Medicine, University of Pennsylvania; Chief of the Department of Diseases of Metabolism, Hospitals of Graduate School of Medicine, University of Pennsylvania; Chief of Department of Metabolism, Philadelphia General Hospital. 1803 Pine St.

1905. Pfahler, George E., M.D., Sc.D., D.M.R.E. (Camb.), Professor of Radiology and Vice-Dean, Department of Radiology, Graduate School of Medicine, University of Pennsylvania; Director of Radiological Department, Misericordia Hospital. 1321 Spruce St.

1915. Pfeiffer, Damon B., A.B., M.D., Assistant Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Surgeon-in-Chief, Abington Memorial and Lankenau Hospitals; Assitant Surgeon, Presbyterian Hospital. 1822 Pine St.

1907. Pfromm, George W., Ph.G., M.D., Assistant Physician to the American Stomach Hospital; Consulting Physician to the German Protestant Home for the Aged. 269 S. Nineteenth St.

ELECTED

1907. Phillips, Horace, M.D., Assistant Surgeon, U. S. Public Health Service; Examining Physician, Office of the District Attorney. 4505 Chester Ave.

1911. Piersol, George Morris, B.S., M.D., Professor of Medicine and Vice-Dean for Medicine, Graduate School of Medicine, University of Pennsylvania; Physician to the Presbyterian and Abington Memorial Hospitals; Active Consultant in Medicine, Philadelphia General Hospital. 1913 Spruce St.

1917. Piper, Edmund Brown, B.S., M.D., Professor of Obstetrics, School of Medicine, University of Pennsylvania; Obstetrician to the University Hospital; Assistant Obstetrician and Gynecologist to the Philadelphia General Hospital; Consulting Obstetrician, Kensington Hospital for Women. 2031 Locust St.

1905. Pitfield, Robert L., M.D., Pathologist to the Germantown Hospital; Bacteriologist to the Chestnut Hill Hospital for Lung Diseases. 5211 Wayne Ave.

1922. Pleasants, Henry, Jr., A.B., M.D., Assistant Visiting Physician, Chester County Hospital, West Chester, Pa.; Assistant in Neurological Clinic, Orthopedic Hospital. West Chester, Pa.

1896. Posey, William Campbell, M.D., Consulting Ophthalmic Surgeon to Wills, Howard, Chestnut Hill and Abington Hospitals. Radnor, Pa.

*1907. Price, George E., M.D. Stimson Building, Seattle, Wash.

1931. Pryor, Charles Allen, M.D., Assistant Oto-laryngologist, Out-Patient Departments of Pennsylvania and Bryn Mawr Hospitals; Assistant in Oto-laryngology, Graduate School of Medicine, University of Pennsylvania. 1910 Spruce St.

*1920. Quicksall, William E., M.D. 222 Taylor Arcade, St. Petersburg, Fla.

1908. Radcliffe, McCluney, A.M. (Lafayette), M.D., LL.D., Consulting Ophthalmologist, Presbyterian Hospital; Consulting Surgeon to the Wills Hospital. 1930 Chestnut St.

1931. Ramsey, Frank M., B.S., M.D., Assistant Attending Physician, Germantown Hospital; Attending Physician, Chestnut Hill Hospital. Chestnut Hill P. O.

1913. Randall, Alexander, A.M., M.D., Associate Professor of Surgery in charge of the Department of Urology, School of Medicine, University of Pennsylvania. 1323 Medical Arts Building.

1887. Randall, B. Alex., M.A., M.D., Ph.D., Emeritus Professor of Otology in the School of Medicine, University of Pennsyl-

ELECTED

vania; Ear Surgeon to the Children's Hospital; Consulting Aurist to the Pennsylvania Institution for the Deaf and Dumb and to Memorial Hospital, Roxborough. 2320 DeLancey St.

1926. Raydin, I. S., B.S., M.D., J. William White Professor of Surgical Research in the School of Medicine and Assistant Professor of Research Surgery in the Graduate School of Medicine, University of Pennsylvania; Surgeon, University Hospital. 4623 Larchwood Ave.

*1904. Ravenel, Mazyck P., M.D., Professor of Preventive Medicine and Bacteriology and Director of the Public Health Laboratory at the University of Missouri. 203 Westmount Ave., Columbia, Mo.

1928. Reese, Warren S., M.D., Assistant Ophthalmologist, Pennsylvania Hospital; Assistant Surgeon, Wills Hospital; Associate Ophthalmologist, Delaware County Hospital; Associate in Ophthalmology, Graduate School of Medicine, University of Pennsylvania. 230 S. Twenty-first St.

1925. Reeves, Rufus S., B.S., M.D. 2227 Spruce St.

1931. Register, Robert P., M.D., Instructor in Medicine, Jefferson Medical College; Physician to Pennsylvania Hospital; Director Medical Science, Delaware County Hospital. 255 S. Sixteenth St.

1920. Rehfuss, Martin E., M.D. N. E. Cor. Sixteenth and Spruce Streets.

1921. Reiff, E. Paul, A.B., M.D., Member of Medical Staff, Methodist Episcopal Hospital. 5051 Chestnut St.

1919. Reimann, Stanley P., M.D., Director of the Research Institute, Lankenau Hospital; Associate Professor of Surgical Pathology, Graduate School of Medicine, University of Pennsylvania. 516 Arbutus St., Germantown.

1929. de Rényi, George S., M.D., Associate Professor of Anatomy, University of Pennsylvania. 216 S. Forty-sixth St.

1923. Repplier, Sidney J., M.D., Medical Director, Curtis Hospital, Curtis Publishing Company. Chestnut Hill, Phila.

†1906. Rhein, Robert D., M.D., Chief Physician to the Clinic of the American Hospital for Diseases of the Stomach; Physician to the Philadelphia Home for Incurables; Examining Physician to the White Haven Sanatorium. S. W. Cor. Fifteenth and Pine Sts.

1919. Richardson, Russell, A.M., M.D., Associate in Medicine, University of Pennsylvania and Cox Medical Research Institute, University of Pennsylvania; Director of the Laboratory, Methodist Episcopal Hospital. 320 S. Sixteenth St.

ELECTED

1919. Ridpath, Robert F., M.D., Professor of Rhino-laryngology, Temple University; Associate Professor of Laryngology, Graduate School of Medicine, University of Pennsylvania; Laryngologist, Samaritan and Jewish Hospitals. 1737 Chestnut St.

1898. Riesman, David, M.D., Sc.D., Professor of Clinical Medicine, School of Medicine, University of Pennsylvania; Visiting Physician to the Philadelphia General and University Hospitals; Consulting Physician to the Jewish, Woman's and Kensington Hospitals. 1520 Spruce St.

1895. Ring, G. Oram, A.M., M.D., Consulting Ophthalmologist to the Episcopal Hospital; Ophthalmologist to the Widener Memorial Home for Crippled Children; Consulting Ophthalmologist to the American Oncologic Hospital. N. E. Cor. Seventeenth and Walnut Sts.

1899. Roberts, Walter, M.D., Professor of Otology in the Graduate School of Medicine, University of Pennsylvania; Otologist to the Methodist Episcopal Hospital; Laryngologist to the Philadelphia General Hospital. 1921 Spruce St.

1903. Robertson, William Egbert, M.D., Professor of Theory and Practice of Medicine and of Clinical Medicine in Temple University; Physician to the Samaritan and Garretson Hospitals. 327 S. Seventeenth St.

1912. Rodman, John Stewart, M.D., Professor of Surgery, Woman's Medical College; Surgeon-in-Chief, Woman's College Hospital; Attending Surgeon, Bryn Mawr Hospital; Assistant Surgeon to the Presbyterian Hospital. 1310 Medical Arts Building.

1928. Rose, Edward, M.D., Instructor in Medicine, University of Pennsylvania; Medical Registrar, University Hospital. 1726 Pine St.

1909. Rosenberger, Randle C., M.D., Professor of Preventive Medicine and Bacteriology in the Jefferson Medical College. 1001 Walnut St.

*1907. Royer, B. Franklin, M.D. 450 Seventh Ave., New York City.

†1905. Rugh, James Torrance, A.B., M.D., LL.D., James Edwards Professor of Orthopedic Surgery, Jefferson Medical College; Orthopedic Surgeon to the Jefferson Medical College Hospital; Orthopedic Surgeon to the Methodist and the Philadelphia General Hospitals. 911 Medical Arts Building.

1929. Ryan, William John, M.D., Visiting Surgeon to St. Mary's Hospital and to St. Vincent's Hospital for Women and Children. Medical Arts Building.

ELECTED

1905. Sartain, Paul J., A.M., M.D. 2006 Walnut St.

1908. Sautter, Albert C., M.D., Assistant in the Eye Dispensary of the Lankenau Hospital. Germantown Professional Building.

1927. Saxon, Gordon J., M.D. 1506 Medical Arts Building.

1906. Saylor, Edwin S., M.D., Consulting Ophthalmologist, American Hospital for Diseases of the Stomach. 1737 Chestnut St.

1920. Scarlett, Hunter W., M.D., Associate in Ophthalmology in the Graduate School of Medicine, and Medical Examiner in the Department of Physical Education, University of Pennsylvania. 230 S. Twenty-first St.

1917. Schaeffer, Jacob Parsons, A.M., M.D., Ph.D., Sc.D., Professor of Anatomy and Director of the Daniel Baugh Institute of Anatomy of the Jefferson Medical College. 4634 Spruce St.

1899. Schamberg, Jay F., M.D., Professor of Dermatology and Syphilology, Graduate School of Medicine, University of Pennsylvania; Director, Research Institute of Cutaneous Medicine. 1402 Spruce St.

1929. Scheffey, Lewis C., P.D., M.D., Assistant Gynecologist, Jefferson Hospital; Associate in Gynecology, Jefferson Medical College. 267 S. Nineteenth St.

1929. Schenck, Harry P., B.S., M.D., Research Fellow in Allergy and Assistant Instructor in Otolaryngology, University of Pennsylvania; Lecturer and Instructor in Pathology, Evans Institute; Assistant Otolaryngologist, Children's Hospital. 1912 Spruce St.

1917. Schnabel, Truman Gross, A.B. (Lehigh), M.D., Associate in Medicine, University of Pennsylvania; Physician, Philadelphia General and Medical Dispensary, University Hospital. 1704 Pine St.

1917. Schoff, Charles H., M.D. Media, Pa.

1928. Schofield, Frederick S., A.B., M.D., Associate in Urology, Medical School, University of Pennsylvania; Chief of Urological Dispensary, University Hospital; Urologist, Orthopedic Hospital; Assistant Urologist, Chestnut Hill Hospital. 822 Medical Arts Building.

1921. Schumann, Edward Armin, A.B., M.D., Associate Professor of Obstetrics, School of Medicine, University of Pennsylvania; Surgeon-in-Chief, Kensington Hospital for Women, Obstetrician and Gynecologist, Philadelphia General and Memorial Hospitals; Obstetrician, Chestnut Hill Hospital. 1814 Spruce Street.

ELECTED

1887. DE SCHWEINITZ, GEORGE EDMUND, A.M., M.D., LL.D., L.H.D., Sc.D., Honorary Professor of Ophthalmology in the Graduate School of Medicine, University of Pennsylvania; Consulting Ophthalmic Surgeon to the Philadelphia General, Bryn Mawr and Chestnut Hill Hospitals; Trustee of the University of Pennsylvania and Honorary Chairman of the Board of Medical Affairs. 1705 Walnut St.

*1913. DE SCHWEINITZ, GEORGE LORD, B.S., M.D., Assistant in the Department of Ophthalmology, St. Luke's Hospital, Bethlehem, Pa. 85 East Broad St., Bethlehem, Pa.

1910. SCHWENK, PETER N. K., M.A., M.D., Attending Surgeon to Eye Department of the Pennsylvania Hospital; Attending Surgeon to the Wills Hospital. 1417 N. Broad St.

1930. SCOTT, JOHN P., M.D., Associate in Pediatrics, University of Pennsylvania; Pediatrician, Presbyterian Hospital; Assistant Physician, Children's Hospital. 2040 Pine St.

1917. SERVICE, CHARLES A., M.D. Bala, Pa.

1924. SHALLOW, THOMAS A., M.D., Professor of Surgery, Jefferson Medical College. 3942 Chestnut St.

1908. SHANNON, CHARLES E. G., A.B., M.D., Instructor in Ophthalmology in the Jefferson Medical College; Assistant in the Ophthalmological Clinic at the Jefferson Medical College Hospital; Ophthalmologist to the Seybert Institution. 1930 Chestnut St.

1928. SHARPE, JOHN SEMPLE, M.D., Attending Physician, Bryn Mawr Hospital. Haverford, Pa.

1928. SHARPLESS, FREDERICK COPE, A.B., M.D., Attending Physician, Bryn Mawr, Hospital. Rosemont, Pa.

1897. SHARPLESS, WILLIAM T., M.D., Physician to the Chester County Hospital. West Chester, Pa.

1931. SHEEHAN, WILLIAM C., M.D., 8820 Germantown Ave.

1906. SHIELDS, WILLIAM G., M.D., Dermatologist to Germantown Hospital; Chief of Dermatological Clinic and Assistant Physician to the Jewish Hospital. 414 School Lane, Germantown.

*1908. SHOEMAKER, HARLAN, A.B., M.D., Chief Surgeon to Los Angeles County Hospital; Chief of the Surgical Staff of the Methodist Hospital and of the Hospital of the Good Samaritan. 1930 Wilshire Building, Los Angeles, Calif.

†1896. SHOEMAKER, WILLIAM T., M.D., Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Clinical Professor of Ophthalmology in the Woman's Medical College of Pennsylvania; Ophthalmic Surgeon to Lankenau, Germantown and Pennsylvania Hospitals. 2017 Locust St.

ELECTED

1900. Shumway, Edward Adams, B.S., M.D., Ophthalmic Surgeon to the Lankenau and Oncologic Hospitals. 1737 Chestnut St.

1903. Sinclair, John Falconer, M.D., Epidemiologist, Presbyterian Hospital; Physician to the Philadelphia Orphan Asylum and to the Presbyterian Orphanage; Physician to the Home of the Merciful Saviour for Crippled Children. 4103 Walnut St.

1907. Sinkler, Francis Wharton, A.B., M.D., Physician to the Orthopedic Hospital and Infirmary for Nervous Diseases. 1226 Medical Arts Building.

1902. Siter, E. Hollingsworth, M.D., Genito-urinary Surgeon to the Philadelphia General Hospital. 2213 DeLancey St.

1928. Skillern, Samuel Ruff, M.D., Assistant Professor in Oto-laryngology, Graduate School of Medicine, University of Pennsylvania. 1734 Pine St.

1929. Smith, Austin Thomas, M.D., Clinical Assistant, Department of Laryngology, Jefferson Hospital; Instructor in Laryngology, Jefferson Medical College. 259 S. Seventeenth St.

1927. Smyth, Calvin M., Jr., B.S., M.D., Assistant Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Surgeon in charge, Methodist Hospital; Visiting Surgeon, Abington Memorial Hospital. 2021 Spruce St.

1919. Smyth, Henry Field, M.D., Assistant Professor of Industrial Hygiene, School of Hygiene, University of Pennsylvania. Wayne, Pa.

1928. Spaeth, Edmund B., M.D., Associate in Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Instructor in Ophthalmology, School of Medicine, Temple University. 1930 Chestnut St.

1908. Speese, John, M.D., Associate Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Surgeon to the Children's and Presbyterian Hospitals. 133 S. Thirty-sixth St.

1897. Spiller, William G., M.D., Professor of Neurology in the University of Pennsylvania; Consulting Neurologist to the Philadelphia General Hospital. 4409 Pine St.

1929. Sponsler, Marshall B., A.B., M.D., Instructor in Oto-laryngology, School of Medicine and Associate in Oto-laryngology, Graduate School of Medicine, University of Pennsylvania; Assistant Oto-laryngologist, Methodist Hospital; Visiting Oto-laryngologist, White Haven Sanatorium. 1934 Spruce St.

1894. Stahl, B. Franklin, B.S., Ph.G., M.D. Haverford, Pa.

1909. Starbuck, J. Clinton, M.D., Physician to the Orphanage, Orphan Society of Philadelphia, at Wallingford, and to the

ELECTED

Glen Mills School, Glen Mills, Pa. 42 E. Washington St., Media, Pa.

1927. Starr, Isaac, Jr., B.S., M.D., Assistant Professor of Clinical Pharmacology and Associate in Medicine, School of Medicine, University of Pennsylvania; Acting Physician, University Hospital. Chestnut Hill.

1912. Stauffer, Nathan Pennypacker, D.D.S., M.D., Laryngologist and Otologist to the Presbyterian Hospital; to the Philadelphia General Hospital, Tubercular Department; to the Home for Crippled Children. 1900 Rittenhouse Square.

1910. Stellwagen, Thomas C., Jr., M.D., Chief Clinical Assistant in the Out-Patient Surgical Department of the Jefferson Medical College Hospital. 220 S. Sixteenth St.

1895. Stengel, Alfred, M.D., Sc.D., Professor of Medicine in the University of Pennsylvania; Physician to the University Hospital and the Pennsylvania Hospital. University Hospital.

1901. Stevens, Arthur A., M.D., Professor of Applied Therapeutics in the University of Pennsylvania; Professor of Materia Medica, Therapeutics and Clinical Medicine in the Woman's Medical College of Pennsylvania; Physician to St. Agnes' Hospital. 314 S. Sixteenth St.

1914. Stewart, Thomas S., B.S. (Univ. of Penna.), M.D. 1532 Pine St.

1931. Stiles, Merritt H., M.D., Assistant in Medicine, in charge Thyroid Dispensary, Germantown Hospital; Assistant in Pediatrics, Chestnut Hill Hospital. 113 W. Chestnut Ave., Chestnut Hill.

1925. Stokes, John H., A.B., M.D., Professor of Dermatology and Syphilology, School of Medicine, and Graduate School of Medicine, University of Pennsylvania; Attending Dermatologist, University and Philadelphia General Hospitals; Dermatologist, Hospital of the Graduate School of Medicine. 3800 Chestnut St.

1928. Stokes, Joseph, Jr., A.B., M.D., William H. Bennett Assistant Professor of Pediatrics, School of Medicine, University of Pennsylvania. 159 W. Coulter St., Germantown.

1898. Stout, George C., M.D., Consulting Laryngologist and Aurist to the Presbyterian Hospital, the Children's Aid Society and the William Penn Charter School. 2015 Walnut St.

1923. Strecker, Edward A., A.M., M.D., Professor of Psychiatry and Chairman of the Department of Psychiatry, School of Medicine, University of Pennsylvania; Clinical Professor of Psychiatry and Mental Hygiene, Yale University Medical

ELECTED

School; Neurologist, Pennsylvania Hospital; Medical Director of Department for Mental and Nervous Diseases, Pennsylvania Hospital. 111 N. Forty-ninth St.

1923. Stroud, William D., B.S., M.D., Physician to the Pennsylvania Hospital and Chief of Heart Clinics; Professor of Cardiology, Graduate School of Medicine, University of Pennsylvania; Associate in Medicine, School of Medicine, University of Pennsylvania; Physician in Charge of the Children's Heart Hospital of Philadelphia. 1011 Clinton St.

1927. Sturgis, Samuel B., M.D. Ardmore, Pa.

1930. Sunderman, F. William, B.S., M.D., M.S., Ph.D., Assistant Professor, Research Medicine, University of Pennsylvania; Assistant Physician and Chemist, Pennsylvania Hospital; Chief of the Diabetic Clinic, Pennsylvania Hospital. 2015 Spruce St.

*1900. Swan, John M., M.D., Attending Physician, Park Avenue Hospital. 457 Park Avenue, Rochester, N. Y.

1928. Swartley, William B., M.D , Surgeon to Germantown Dispensary and Hospital and to Chestnut Hill Hospital; Demonstrator of Anatomy, Jefferson Medical College. 6002 Greene St., Germantown.

1900. Talley, James Ely, A.B., M.D., Professor of Cardiology, Graduate School of Medicine, University of Pennsylvania; Consulting Physician to the Presbyterian Hospital. 1811 Spruce St.

‡1886. Taylor, John Madison, A.B., and A.M. (Princeton), M.D., Professor of Physical Therapeutics and Dietetics, Medical Department, Temple University. 1504 Pine St.

1931. Taylor, Norman H., A.B., M.A., M.D., Associate Pediatrist, Presbyterian Hospital; Assistant Pediatrist, Chestnut Hill Hospital. 100 W. Evergreen Ave.

1887. Taylor, William J., M.D., Surgeon to the Orthopedic Hospital and Infirmary for Nervous Diseases; Consulting Surgeon to St. Agnes', Woman's and Abington Memorial Hospitals. 1825 Pine St.

‡1886. Taylor, William L., M.D. 2223 Tioga St.

1928. Thomas, J. Earl, M.D. Drexel Hill, Pa.

1907. Thomas, Thomas Turner, M.D., Associate Professor of Applied Anatomy and Associate in Surgery in the University of Pennsylvania; Surgeon to the Philadelphia General Hospital; Assistant Surgeon to the University Hospital. 1737 Chestnut Street.

ELECTED

1927. Thorington, J. Monroe, B.S., A.M., M.D., Instructor in Ophthalmology, University of Pennsylvania; Assistant in Ophthalmology, Presbyterian Hospital. 2031 Chestnut St.

1898. Thornton, Edward Q., M.D., Assistant Professor of Materia Medica in the Jefferson Medical College. 1331 Pine St.

1930. Thorpe, Edward S., Jr., B.S., M.D., Associate in Pediatries and Assistant to the Dean, School of Medicine, University of Pennsylvania; Visiting Physician to Children's, University and Philadelphia General Hospitals. 133 S. Thirty-sixth St.

1912. Torrey, Robert G., M.D., Professor of Principles and Practice of Medicine, Woman's Medical College; Associate in Medicine, School of Medicine, University of Pennsylvania; Physician to the Philadelphia General Hospital. 1716 Locust St.

1908. Tracy, Stephen E., M.D., Gynecologist to the Stetson Hospital; Visiting Surgeon to the Gynecean Hospital. 1930 Chestnut St.

1926. Tucker, Gabriel, M.D., Associate Professor of Bronchoscopy and Esophagoscopy, Graduate School of Medicine, and Associate Bronchoscopic Surgeon, University Hospital, University of Pennsylvania; Associate in Bronchoscopy and Assistant Bronchoscopist, Jefferson Medical College and Hospital; Bronchoscopist, Misericordia and Bryn Mawr Hospitals. 326 S. Nineteenth St.

*†1901. Tucker, Henry, M.D. Bellevue, Md.

†1894. Tunis, Joseph Price, M.D. University Club, Philadelphia.

1923. Turner, Creighton H., M.D., Associate in Medicine, Jefferson Medical College; Visiting Physician, St. Agnes' Hospital. 1731 Pine St.

1929. Tyson, Ralph M., M.D., Associate in Pediatries, Jefferson Medical College; Pediatrist, Pennsylvania Hospital. 334 S. Twenty-first St.

1907. Ullom, Josephus Tucker, M.A., M.D., Physician to the Chestnut Hill Hospital. Carpenter and Quincy Streets, Germantown.

*1913. Vail, William Penn, B.S., M.S., M.D., Laryngologist to the Pennsylvania Institution for the Instruction of the Blind; Laryngologist to the Department for Tuberculosis of the Philadelphia General Hospital; Assistant Laryngologist and Otologist to the Out-Patient Department of the Pennsylvania Hospital; Assistant Laryngologist to the Children's Hospital. Flairstown, N. J.

ELECTED

1873. Van Harlingen, Arthur, Ph.B. (Yale), M.D., Emeritus Professor of Diseases of the Skin in the Philadelphia Polyclinic. Bryn Mawr, Pa.

1903. Van Pelt, William Turner, M.D. Chestnut Hill, Philadelphia.

1912. Vaux, Norris Wistar, M.D., Clinical Professor of Obstetrics, Jefferson Medical College; Chief of Maternity Service, Philadelphia Lying-in and Pennsylvania Hospitals; Surgeon to the Chestnut Hill Hospital; Surgeon to the Dispensary of the Germantown Hospital. 2031 Locust St.

*1897. Veasey, Clarence A., A.M., M.D., Ophthalmic Surgeon to the Deaconess and to St. Luke's Hospitals, Spokane. Suite 1569, Paulsen Medical and Dental Building, Spokane, Wash.

‡1883. Vinton, Charles Harrod, A.M., M.D. 1300 Locust St.

1903. Wadsworth, William Scott, M.D. 3914 Baltimore Ave.

1927. Wagers, Arthur J., M.D., Associate in Otology, Jefferson Medical College; Demonstrator of Laryngology, Jefferson Medical College; Otologist, Joseph Price Hospital. 1429 Spruce St.

1928. Wagoner, George W., M.D., Assistant Professor of Orthopedic Surgery, Graduate School of Medicine, and Associate in Pathology, School of Medicine, University of Pennsylvania; Clinical Surgeon, Orthopedic Hospital; Orthopedic Surgeon, Bryn Mawr Hospital. 2008 Walnut St.

1931. Walker, Herschel C., M.D., Assistant Surgeon, University Hospital; Instructor in Urology, School of Medicine, University of Pennsylvania and Woman's Medical College. 1323 Medical Arts Building.

1906. Walker, John K., M.D., Physician to the Children's Hospital of the Mary J. Drexel Home; Physician to the Out-Patient Department of the Pennsylvania Hospital. Media, Pa.

1907. Walker, Warren, M.D., Dermatologist to the Episcopal and Bryn Mawr Hospitals. 1819 Chestnut St.

1904. Walsh, Joseph, A.M., M.D., Visiting Physician to and Medical Director of the White Haven Sanatorium. Medical Arts Building.

1895. Watson, Arthur W., M.D., Professor of Diseases of the Throat and Nose in the Philadelphia Polyclinic and College for Graduates in Medicine; Laryngologist to the Jewish Hospital; Laryngologist and Aurist to the Mt. Sinai Hospital; Laryngologist to Home for Incurables. 2013 Locust St.

ELECTED

1923. Watson, William R., M.D., Chief Laryngologist, Episcopal and Diagnostic Hospitals; Assistant Chief Laryngologist, Pennsylvania Hospital. 2124 Pine St.

1903. Weber, Charles H., M.D., Assistant Professor of Pediatrics, Graduate School of Medicine, University of Pennsylvania; Consulting Physician to the Medical Dispensary, Children's Hospital; Pediatrist to the Municipal Court. 2048 Pine St.

1930. Weeder, S. Dana, M.D., Assistant Surgeon, Germantown and Chestnut Hill Hospitals; Instructor in Anatomy, School of Medicine, University of Pennsylvania. 6110 Greene St., Germantown.

1920. Weidman, Fred. D., M.D., Professor of Dermatological Research, University of Pennsylvania; Assistant Pathologist, Philadelphia Zoölogical Garden; Attending Dermatologist, Philadelphia General Hospital. 20 Tenby Road, Llanerch, Pa.

1906. Weisenburg, Theodore H., M.D., Professor of Neuro-Psychiatry, Graduate School of Medicine, University of Pennsylvania; Neurologist, Philadelphia General Hospital, and Physician to Orthopedic Hospital and Infirmary for Nervous Diseases. 1930 Chestnut St.

1923. Weiss, Edward, M.D., Associate in Medicine and Pathology, Jefferson Medical College; Physician to the Tuberculosis Wards, Philadelphia General Hospital. 1923 Spruce St.

1913. Wells, P. Frailey, A.M., M.D., Ph.D. 754 N. Fortieth St.

1893. Westcott, Thompson S., M.D., Consulting Pediatrist to the Methodist Episcopal Hospital; Consulting Pediatrist, Haddock Memorial Home. 1720 Pine St.

1921. Whitaker, William, M.D. 5448 Germantown Ave.

1901. White, Courtland Y., M.D., Director of the Pathological Laboratories of the Episcopal Hospital; Pathologist to the Children's Hospital and to the Kensington Hospital for Women; Chief Bacteriologist to the Bureau of Health, Department of Health and Charities, Philadelphia; Associate Professor of Bacteriology in the Graduate School of Medicine, University of Pennsylvania. 6611 N. Tenth St.

*1905. Whiteway, Harold M., M.D. 2356 W. Twenty-first St., Los Angeles, Calif.

1898. Whiting, Albert D., M.D., Associate Professor of Surgery, Graduate School of Medicine, University of Pennsylvania; Associate Surgeon, Lankenau Hospital; Surgeon, Germantown Hospital. 333 S. Eighteenth St.

1925. Widmann, Bernard P., M.D., Assistant Professor of Radiology, Graduate School of Medicine, University of Pennsylvania;

ELECTED

Chief of the Radiological Department of the Philadelphia General Hospital. 250 S. Eighteenth St.

1914. Willard, de Forest P., B.S. (Univ. of Penna.), M.D., Professor of Orthopedics, Graduate School of Medicine, University of Pennsylvania; Orthopedic Surgeon to the Graduate Orthopedic and Delaware Hospitals. 1916 Spruce St.

1907. Williams, Carl, B.S., M.D., Ophthalmic Surgeon to the Germantown Hospital; Instructor in Ophthalmology in the University of Pennsylvania; Ophthalmic Surgeon to the Pennsylvania Institution for the Deaf and Dumb. School Lane and Greene Sts., Germantown.

1924. Williams, Horace James, M.D., Otolaryngologist to Germantown, Roxborough Memorial and Municipal Hospitals; Instructor in Otolaryngology, School of Medicine, University of Pennsylvania. 5908 Greene St., Germantown.

1920. Williams, Philip F., Ph.B., M.D., Assistant Professor of Obstetrics, School of Medicine and Graduate School of Medicine, University of Pennsylvania; Gynecologist and Obstetrician to Philadelphia and Jewish Hospitals; Obstetrician, Abington Memorial Hospital. 2206 Locust St.

1927. Williamson, Ernest G., M.D., Surgeon, Children's Hospital; Assistant House Surgeon, Pennsylvania Hospital; Assistant Surgeon, Presbyterian and Jefferson (Dispensary) Hospitals. 6353 Woodbine Ave.

1916. Wilmer, Harry B., M.D., Assistant Professor of Clinical Allergy in the Graduate School of Medicine, University of Pennsylvania; Visiting Physician to the Germantown Hospital; Associate Physician, Presbyterian Hospital; Director of the Allergic Clinic, Germantown and Presbyterian Hospitals. 138 W. Walnut Lane, Germantown.

1921. Wilson, George, M.D., Assistant Professor of Neurology, School of Medicine, University of Pennsylvania; Clinical Professor of Neurology, Women's Medical College of Pennsylvania; Neurologist, Philadelphia General, Episcopal and Abington Memorial Hospitals; Assistant Neurologist, University Hospital. 133 S. Thirty-sixth St.

1874. Wilson, James Cornelius, A.M. (Princeton), M.D., Emeritus Professor of the Practice of Medicine and of Clinical Medicine in the Jefferson Medical College; Physician-in-Chief (Emeritus) to the Lankenau Hospital; Emeritus Physician to the Pennsylvania Hospital; Consulting Physician to the Bryn Mawr Hospital. 1509 Walnut St.

1902. Wilson, Samuel M., M.D. 1930 Chestnut St.

1897. Wilson, W. Reynolds, M.D. Villa Nova, Pa.

ELECTED

1904. Wister, James W., M.D., Physician to the Out-Patient Department of the Germantown Hospital. 5430 Germantown Ave.

*1901. Witmer, A. Ferree, M.D. 21 North Grand Ave., Baldwin, N. Y.

1918. Wolferth, Charles Christian, A.B., M.D., Assistant Professor of Medicine, University of Pennsylvania; Director of the Clinic, Robinette Foundation, University Hospital; Consulting Cardiologist, Jewish Hospital. S. E. Cor. Thirty-sixth and Spruce Sts.

1893. Wood, Alfred C., M.D., Assistant Professor of Surgery in the University of Pennsylvania; Surgeon to the University, the Philadelphia General and St. Timothy's Hospitals. 2035 Walnut St.

1900. Wood, George B., M.D., Oto-laryngologist, Orthopedic Hospital; Professor of Laryngology, Graduate School of Medicine, University of Pennsylvania. N. E. Cor. Twentieth and Chestnut Sts.

1903. Wood, Horatio C., Jr., M.D., Professor of Pharmacology and Therapeutics in the University of Pennsylvania; Professor of Materia Medica, Philadelphia College of Pharmacy and Science. 319 S. Forty-first St.

*1911. Woods, Andrew H., A.B., M.D., Professor of Psychiatry, College of Medicine, and Director of the Psychopathic Hospital, University of Iowa. 1100 N. Dubuque St., Iowa City, Iowa.

†1897. Woodward, George, M.D. W. Willow Grove Ave., Chestnut Hill, Philadelphia.

1903. Worden, Charles B., A.M., M.D. Princeton, N. J.

1928. Wright, Carroll S., B.Sc., M.D., Professor of Dermatology-Syphilology, Temple University School of Medicine; Associate Professor of Dermatology-Syphilology, Graduate School of Medicine, University of Pennsylvania; Attending Radiologist, Philadelphia General Hospital; Attending Dermatologist, Municipal Hospital. 1402 Spruce St.

1894. Zentmayer, William, M.D., Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania; Consulting Surgeon to the Wills Hospital; Consulting Ophthalmologist to the Glen Mills School and to St. Mary's Hospital. 1930 Chestnut St.

‡1887. Ziegler, Walter M.L., A.M., M.D. 1418 N. Seventeenth St.

1895. Zimmerman, Mason W., M.D., Consulting Ophthalmic Surgeon to the Germantown Hospital. 1518 Waverly St.

1931. Zozaya, José, B.S., M.D., Dr. P.H., Assistant Director of Mulford Biological Laboratories. Devon, Pa.

ASSOCIATE FELLOWS

(Limited to Fifty, of whom Twenty may be Foreigners)

American

ELECTED

1909. Billings, Frank, M.D., 1550 North State Parkway, Chicago, Illinois.

1893. Councilman, William T., M.D., Harvard Medical College, Boston, Massachusetts.

1909. Crile, George W., M.D., 1021 Prospect Avenue, S. E., Cleveland, Ohio.

1909. Dana, Charles Loomis, M.D., 53 West Fifty-third Street, New York City, New York.

1916. Estes, William Lawrence, M.D., 805 Delaware Ave., Bethlehem, Pa.

1931. Garrison, Fielding H., M.D., Washington, D. C.

1912. McCaw, Brig.-Gen. Walter D., M.D., Assistant Surgeon-General, U. S. A., retired, Woodstock, Ulster Co., N. Y.

1906. Mayo, William J., M.D., Rochester, Minnesota.

1917. Miner, Charles H., M.D., Wilkes-Barre, Pa.

1906. Pilcher, Lewis Stephen, M.D., 489 Washington Avenue, Brooklyn, New York.

1892. Welch, William H., M.D. 1900 E. Monument St., Baltimore, Maryland.

Foreign

1928. Archibald, Edward, M.D., McGill University, Montreal, Canada.

1924. Hopkins, Sir F. Gowland, M.D., Biochemical Department, University of Cambridge, Cambridge, England.

1909. Macallum, Archibald B., M.A., M.B., Ph.D., Sc.D., LL.D., F.R.S., Department of Biochemistry, McGill University, Montreal, Canada.

1922. McCarrison, Colonel Robert, M.D., C.I.E., D.Sc. LL.D., F.R.C.P. (London), Honorary Physician to the King. Pasteur Institute, Coonoor, S. India.

ELECTED

1924. Macleod, John James R., M.D., M.B., D.Sc. (Hon.), LL.D., F.R.S., 43 Nanton Ave., Toronto, Canada.

1906. Myles, Sir Thomas, M.D., 33 Merion Square, W. Dublin, Ireland.

1885. Rendu, Jean, M.D., Lyons, France.

1928. Rolleston, Sir Humphry, Bart., G.C.V.O., K.C.B., M.D., D.C.L. (Durh.), LL.D., D.G.C., "Southfield," Trumpington Road, Cambridge, England.

1908. Ross, Col. Sir Ronald, K.C.B., K.C.M.G., F.R.S., M.D., D.Sc., LL.D., Ross Institute and Hospital for Tropical Diseases, Putney Heath, London, S. W. 15, England.

NECROLOGICAL LIST

FELLOWS

DR. HENRY LEFFMANN	December 25, 1930
DR. SAMUEL W. MORTON	January 12, 1931
DR. WILLIAM DUFFIELD ROBINSON	January 24, 1931
DR. HARVEY SHOEMAKER	February 2, 1931
DR. FRANCIS X. DERCUM	April 23, 1931
DR. CHARLES K. MILLS	May 28, 1931
DR. WALTER M. L. ZIEGLER	May 29, 1931
DR. HOBART A. HARE	June 15, 1931
DR. HARRY C. DEAVER	June 25, 1931
DR. L. NAPOLEON BOSTON	July 4, 1931
DR. HORACE H. JENKS	July 5, 1931
DR. GEORGE I. MacLEOD	July 6, 1931
DR. WILLIAM L. TAYLOR	August 7, 1931
DR. CHARLES H. VINTON	August 20, 1931
DR. JOHN B. DEAVER	September 25, 1931
DR. J. MADISON TAYLOR	October 3, 1931

LIST OF PRIZES AND LECTURES

THE WILLIAM F. JENKS MEMORIAL PRIZE

(Triennial)

1889	John Strahan, M.D.	Belfast (Ireland).
1895	Abram Brothers, M.D.	New York.

NOTE.—June 12, 1900, the William F. Jenks Memorial Prize Fund was transferred to the Library as the "William F. Jenks Memorial Library Fund."

ALVARENGA PRIZE OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA

(Annual)

1890	R. W. Philip, M.D.	Edinburgh (Scotland)
1891	L. Duncan Bulkley, M.D.	New York.
1892	R. H. L. Bibb, M.D.	Saltillo.
1894	G. E. de Schweinitz, M.D.	Philadelphia.
1895	Guy Hinsdale, M.D.	Philadelphia.
1897	Joseph Collins, M.D.	New York.
1898	S. A. Knopf, M.D.	New York.
1898	Robert Randolph, M.D.	Baltimore.
1900	David de Beck, M.D.	Cincinnati.
1901	George W. Crile, M.D.	Cleveland.
1903	William S. Carter, M.D.	Galveston.
1905	D. Chalmers Watson, M.D.	Edinburgh (Scotland).
1907	William Louis Chapman, M.D.	Providence.
1908	William T. Shoemaker, M.D.	Philadelphia.
1910	M. Katzenstein, M.D.	Berlin (Germany).
1911	Francis D. Patterson, M.D.	Philadelphia.
1914	H. B. Sheffield, M.D.	New York.
1915	J. E. Sweet, M.D.	Philadelphia.
1917	Wilburt C. Davison, M.D.	Baltimore.
1921	John W. Churchman, M.D.	New York.
1923	Edward P. Heller, M.D.	Kansas City, Mo.
1924	Gordon Cameron, M.B., B.S.	Victoria, Australia.
1925	Raphael Isaacs, M.D.	Boston.
1926	P. S. Pelouze, M.D. and Frederick S. Schofield, M.D.	Philadelphia.
1927	Emil Bogen, M.D.	Cincinnati, Ohio.
1928	J. Rodman Paul, M.D. and William U. McClenahan	Philadelphia.
1929	George M. Dorrance, M.D.	Philadelphia.
1930	Henry A. Harris, M.D.	London (Eng.).
1931	Edgar S. J. King, M.D.	Melbourne (Australia).

NATHAN LEWIS HATFIELD PRIZE FOR ORIGINAL RESEARCH IN MEDICINE

(Triennial)

1901	Henry F. Harris, M.D.	Atlanta.
1909	Martin Henry Fischer, M.D.	Oakland.
1917	A. B. Macallum, M.D., F.R.S.	Toronto.
1919	Harvey Cushing, M.D.	Boston.
1921	William R. Nicholson, M.D.	Philadelphia.
1922	Sir Thomas Lewis, C.B.E., M.D., F.R.S.	London (Eng.).
1923	Frederick Grant Banting, M.D.,	Toronto.
1924	George J. Heuer, M.D.	Cincinnati.
1926	Knud Faber, M.D.	Copenhagen.
1928	John James R. Macleod	Toronto, Can.
1929	Simon Flexner, M.D.	New York.
1930	Franz Alexander	Berlin, Germany.

NOTE.—November 29, 1913, by Supplemental Deed of Trust, the title of this Fund was changed to "Nathan Lewis Hatfield Prize and Lectureship."

WEIR MITCHELL LECTURES

Jan. 17, 1911	Arthur R. Cushny, M.D.	London.
Mar. 30, 1911	Edmund B. Wilson, Ph.D., LL.D.	New York.
May 16, 1911	Svante Arrhenius	Stockholm.
Nov. 3, 1911	William T. Porter, M.D.	Boston.
Mar. 29, 1912	William H. Howell, M.D.	Baltimore.
Oct. 21, 1912	G. H. F. Nuttall, F.R.S., M.D.	Cambridge (Eng.).
April 4, 1913	H. P. Armsby, Ph.D., LL.D.	Pennsylvania.
Feb. 25, 1914	Harvey Cushing, M.D.	Boston.

PUBLIC LECTURES

Feb. 16, 1910	S. Weir Mitchell, M.D.	Philadelphia.
Nov. 17, 1910	Simon Flexner, M.D.	New York.
Dec. 15, 1910	William H. Welch, M.D.	Baltimore.
April 18, 1911	James G. Mumford, M.D.	Clifton Springs, N. Y.
Nov. 20, 1911	Talcott Williams, A.M., LL.D., Litt.D.	Philadelphia.
April 29, 1912	Owen Wister, A.M., LL.D.	Philadelphia.
Feb. 17, 1913	John K. Mitchell, M.D.	Philadelphia.
Feb. 15, 1916	Daniel J. McCarthy, M.D., and Walter Estell Lee, M.D.	Philadelphia.
April 15, 1916	Surgeon A. M. Fauntleroy	U. S. Navy.

MARY SCOTT NEWBOLD LECTURES

Mar. 29, 1917	A. T. McCormack, M.D.	Bowling Green, Ky.
April 4, 1919	Col. Thomas W. Salmon, M.C.,	U. S. Army.
Feb. 6, 1920	Alonzo E. Taylor, M.D.	Philadelphia.
Feb. 4, 1921	Maj.-Gen. Merritte W. Ireland	Washington.
April 20, 1921	Prof. William Romaine Newbold,	Philadelphia.
Nov. 11, 1921	Robert McCarrison, M.D., D.Sc., LL.D.	Oxford (Eng.).
Feb. 1, 1922	Hans Zinsser, M.D.	New York.
April 5, 1922	G. Canby Robinson, M.D.	Baltimore.
Dec. 6, 1922	Donald C. Balfour, M.D.	Rochester, Minn.
Mar. 7, 1923	J. J. R. Macleod, M.D.	Toronto.
April 4, 1923	Prof. Leon Asher, M.D.	Berne (Switzerl'd).
Mar. 5, 1924	Prof. A. Biedl	Prague (Czecho-Slovakia)
April 15, 1924	J. G. FitzGerald, M.D.	Toronto.
Oct. 31, 1924	John I. Hunter, M.D., and N. D. Royle, M.D.	Sydney (Australia).
Dec. 4, 1924	Willem Einthoven, M.D.	Leyden (Holland).
April 21, 1925	A. N. Richards, A.M., Ph.D.	Philadelphia.
Feb. 2, 1927	Eugene L. Opie, M.D.	Philadelphia.
Nov. 2, 1927	George R. Minot	Boston.
Mar. 22, 1928	Sir Humphry Rolleston, Bart., M.D.	Cambridge (Eng.).
Nov. 7, 1928	Ralph Pemberton, M.D.	Philadelphia.
Oct. 2, 1929	Joseph Barcroft, C.B.E., F.R.S.	Cambridge, (Eng.).
April 22, 1930	Sir Wilfred Grenfell, K.C.M.G., M.D.	Labrador.
May 7, 1930	Charles Singer, M.D., F.R.C.P.	London (Eng.).
Nov. 5, 1930	Prof. M. Weinberg	Paris (France).
Feb. 4, 1931	W. J. Merle Scott, M.D.	Rochester, N. Y.
Mar. 4, 1931	George Burgess Magrath, M.D.	Boston.
Oct. 28, 1931	Sir Thomas Leurs, M.D.	London (Eng.).
Nov. 4, 1931	F. J. Lang, M.D.	Innsbruck (Austria)

MÜTTER LECTURES

1865	J. H. Packard, M.D.	Philadelphia.
1866	J. H. Packard, M.D.	Philadelphia.
1867	J. H. Packard, M.D.	Philadelphia.
1868	Harrison Allen, M.D.	Philadelphia.
1869	J. H. Brinton, M.D.	Philadelphia.
1872	J. da S. Solis Cohen, M.D.	Philadelphia.
1879	S. W. Gross, M.D.	Philadelphia.
1882	E. O. Shakespeare, M.D.	Philadelphia.
1885	H. F. Formad, M.D.	Philadelphia.

1888	O. H. Allis, M.D.	Philadelphia.
1890–1891	Roswell Park, M.D.	Buffalo.
1893–1894	De Forest Willard, M.D., and Guy Hinsdale, M.D.	Philadelphia.
1896	O. H. Allis, M.D.	Philadelphia.
1899–1900	J. B. Roberts, M.D.	Philadelphia.
1901	H. W. Cushing, M.D.	Boston.
1902	L. A. La Garde, M.D.	Washington.
1903	C. N. B. Camac, M.D.	New York.
1904	G. H. Monks, M.D.	Boston.
1905	A. O. J. Kelly, M.D.	Philadelphia.
1906	W. J. Mayo, M.D.	Rochester, Minn.
1907	J. Rogers, M.D., and S. P. Beebe, M.D.	New York.
1908	G. W. Crile, M.D.	Cleveland.
1909	H. D. Fry, M.D.	Washington.
1910	T. W. Hastings, M.D.	New York.
1911	C. F. Nassau, M.D.	Philadelphia.
1912	J. C. Bloodgood, M.D.	Baltimore.
1913	R. C. Coffey, M.D.	Portland, Ore.
1914	F. H. Albee, M.D.	New York.
1915–1916	Rudolph Matas, M.D.	New Orleans, La.
1916	Nelson M. Percy, M.D.	Chicago, Ill.
1917	Chevalier Jackson, M.D.	Philadelphia.
1919	Alexis V. Moschcowitz, M.D.	New York.
1920	J. Chalmers Da Costa, M.D.	Philadelphia.
1921	Prof. H. C. Jacobaeus	Stockholm (Swed.).
1922	James Ewing, M.D.	New York.
1923	J. Edwin Sweet, M.D.	Philadelphia.
1924	Evarts Graham, M.D.	St. Louis.
1925	Edward Archibald, M.D.	Montreal, Can.
1926	Walton Martin, M.D.	Ithaca, N. Y.
1927	E. Starr Judd, M.D.	Rochester, Minn.
1928	Herbert W. Carson, M.D.	London (Eng.).
1929	Robert B. Greenough, M.D.	Boston.
1930	Frank H. Lahey, M.D.	Boston.
1931	Edwin Beer, M.D.	New York.

WEIR MITCHELL ORATION

1919	Charles W. Burr, M.D.	Philadelphia.
1922	Charles Loomis Dana, M.D.	New York.
1925	Simon Flexner, M.D.	New York.
1928	Frederick Tilney, M.D.	New York.
1931	Francis R. Packard, M.D.	Philadelphia.

MEMOIR OF DR. JOSEPH SEAL NEFF*

By WALTER CHRYSTIE, M.D.

Dr. Joseph Seal Neff was born at Twentieth and Chestnut Streets, Philadelphia, February 7, 1854, nearly seventy-seven years before his death, which occurred at his home at Cynwyd on November 4, 1930.

On leaving school Dr. Neff entered the University of Pennsylvania, from which he was graduated in 1873. From the University he went to the Jefferson Medical College, receiving his degree in 1875, Jefferson at that time, like all other medical schools in this country, requiring but two winters of lectures and clinics.

In the following year he became a resident physician in the Orthopaedic Hospital, and in 1878 was appointed to a similar position in the Pennsylvania Hospital, serving one year, and in chronological order being the one hundred and twentieth appointee to that much-sought-for position.

Following this training he spent a year doing hospital work in Vienna and Berlin. Returning to Philadelphia, he soon received an appointment as attending physician to the Philadelphia Hospital and also the post of Coroner's physician, at the same time taking care of a growing private practice.

In 1896 he was appointed to a staff position in the Jefferson Hospital, and was also made demonstrator of anatomy in the medical school.

Early in Dr. Neff's career an endocarditis followed by mitral insufficiency handicapped him heavily and finally caused him to give up private practice, which, financially, he was well able to do. But he was not idle He retained his position on the staff of the Philadelphia Hospital and the Jefferson Hospital until 1907, and took an active part in planning the new Jefferson Hospital buildings.

Dr. Neff had shown rare ability as an organizer and executive, and recognizing these qualities Mayor Reyburn, in 1907, appointed him Director of Public Health and Charities, a position carrying with it many responsibilities and new problems.

It was a reform administration and Dr. Neff was unhampered in carrying out many policies of improvement in his department,

* Read, May 11, 1931.

some of which had already been started by his predecessor, Dr. Edward Martin.

He established divisions for the inspection of tenement houses, for the examination of the eyes and teeth of public school children and for the care of sick babies during the summer months. He was also instrumental in having laws passed controlling the condition of the milk supply of Philadelphia, requiring the licensing of midwives and making spitting in public an offense. He organized a corps of nurses for work in the public schools and a course of lectures for social service workers. He organized a municipal laboratory at Holmesburg and one at City Hall. He built a sanatorium at Byberry for tuberculosis and one for mental diseases. Chinatown, which was unbelievably dirty and unhygienic, received a thorough house-cleaning.

In 1911 Mayor Blankenburg succeeded Mayor Reyburn and reappointed Dr. Neff, who served through to the end of the administration in 1914.

Dr. Neff's personal characteristics were marked. Considerably over six feet tall, finely proportioned, a kindly face and twinkling eye, with a courteous, genial manner toward all, he had a host of devoted frinds A hard worker, he also enjoyed companionship and sport, being particularly fond of quail- and duck-shooting and an excellent shot.

During the last two years of increasing cardiac decompensation he never complained, was never irritable or impatient. Every attention of his nurses was met by a courteous "thank you" even when breath was precious.

Dr. Neff was author of the following articles: "Proper Institutional Care of the Feeble Minded," "Epidemiology of Typhoid Fever," "Municipal Control of Tuberculosis," "Organizations and Functions of Health Officers."

He was president of the Child Federation and vice-president of the American Association for the Study and Prevention of Infant Mortality.

He was a member of the Rittenhouse Club, Union League, Philadelphia Country Club, Merion Cricket Club, Philadelphia County Medical Society, American Medical and American Public Health Associations, American Academy of Medicine, Sons of the Revolution, National Committee of One Hundred, American Academy of Social and Political Science, Historical Society, American Association for the Study and Prevention of Infant Mortality.

ANNUAL ADDRESS OF THE PRESIDENT*

By FRANCIS R. PACKARD, M.D.

I am sorry to say that there has been no improvement in the financial status of the College during the past year. The property at Thirteenth and Locust Streets remains unsold and we, therefore, still carry the burden of its taxes. The present lease of the property as an open-air garage expires this May and I am glad to be able to state that a new lease has been arranged whereby there will be a material increase in the rental. The Finance Committee with the aid of the officers and the various committees of the College, has made the most earnest effort to reduce the expenses of the College for the ensuing year. All have cheerfully coöperated to this end.

I shall not refer further to the financial affairs of the College, as those are sufficiently dealt with in the reports of the Committee on Finance and the Treasurer and the statement which has been drawn up by them. There is an amendment to the by-laws before you for action this evening which was postponed for consideration from the business meeting last May. This amendment provides for an increase in the annual contribution. The Special Committee to consider the Financial Condition of the College, which the President was authorized to appoint last year, is unanimously opposed to an increase in the annual contribution at the present time, for obvious reasons. It suggested if this amendment is not adopted that each Fellow be requested to voluntarily add to his annual contribution such sum as he may feel able and inclined to give. Many Fellows have not given anything to the College because they have not felt able to give as large a sum as they thought might be expected. A short time ago your President sent out a letter in an effort to ascertain the feelings of the Fellows in this matter. It met with a most gratifying response. If the amendment does not pass, and those who can would each contribute even a small sum, it would obviate the necessity for imposing a burden on those who are unable to bear it.

* Read, January 11, 1932.

Last spring your President sent out several letters concerning the affairs of the College, not so much with the object of raising money as with the hope of making all the Fellows more familiar with the details of the business of the College and inciting them to take more active interest in it. A great many Fellows were kind enough to take the trouble to answer these communications and many of the replies contained valuable suggestions. I have studied their answers with great care and, though time does not permit me to deal with them all, I am going to refer to some.

First. As regards the Fellowship, many think it should be much larger. This seems to me obvious. Our Active Fellowship this year is 467, the same number as in 1930, and 7 less than it was in 1920. This can be largely overcome if the Fellows would look out for suitable men to propose, and when they have proposed a man take an active interest in trying to have their candidate elected. Too often the proposers each write, as required, an often perfunctory letter, in support of the candidate, and take no pains to give the Council further information. If the Council recommends the candidate his proposers frequently do not attend the meeting of the College when the candidate's name comes up for election, and there have been not a few instances where he has failed to be elected for this reason.

Among the suggestions for increasing the Fellowship was one that the geographical limitations of the Fellowship should be extended, so that physicians living at a distance of more than thirty-five miles from Philadelphia, or in New Jersey, would be eligible. Another suggestion was that the by-laws should be amended so that research workers in medicine and allied subjects, such as chemistry and physics, who had not the degree of M.D., should be eligible. Others suggested that the Fellowship should be divided in various classes, such as Senior and Junior, and one suggestion was the creation of a class of dental Fellows; a great number of the letters urged the election of women to Fellowship.

Second. As to the annual contribution. About an equal number thought it was too high or too low. One suggested grading it, so that some Fellows would agree to pay a larger sum and others less, according to their ability or inclination.

Third. There were words of commendation of the Library administration and some of condemnation. The latter chiefly criticized the Library because of what they considered illiberality

as to the rules regarding the use of the books; and various regulations regarding the reading rooms, especially the manner in which these regulations are enforced.

Your President took the pains to answer each of the letters he received. In most instances the gist of what he said was that any Fellow who felt that any change in the by-laws was necessary had the remedy in his own hands. If he wishes he, with four other Fellows, can present a proposed amendment on the floor of the College, and it will be acted upon in due course. Also, if he would attend the business meetings he could there express his views in the presence of the Fellowship, and I do not doubt they would receive the attention they deserve.

Meetings. During the past year the College held three business meetings and eight scientific meetings. Four lectures were delivered under the Mary Scott Newbold Lecture Fund: on February 4, by Dr. W. D. Merle Scott, of Rochester, N. Y., on "Sympathetic Activity in Certain Diseases, Especially Those of the Peripheral Circulation;" March 4, Dr. George Burgess Magrath, of Boston, spoke on "Medical Science in the Service of the State; Some Professional Experiences;" October 28, Sir Thomas Lewis, of London, on "Vasovagal Syncope and the Carotid Sinus Mechanism;" November 4, Dr. F. J. Lang, of Innspruck, Austria, on "Osteoarthritis Deformans Contrasted With Osteoarthritis Deformans Juvenilis."

On April 1, Dr. C. E. A. Winslow, of Boston, delivered an address on "The Modern Community Health Programme," under the auspices of the James M. Anders Fund.

The Mütter Lecture was delivered on November 4, by Dr. Edwin Beer, of New York, on "Diverticulosis of the Alimentary and Urinary Tract."

The fifth Weir Mitchell Oration was delivered by Dr. Francis R. Packard on November 18, on "How Medicine in Philadelphia was Influenced by London and Edinburgh in the Eighteenth Century."

The Library. The total number of volumes in the Library is now 166,706. We have 400 Incunabula and 440 medical manuscripts. During the past year the Library has been kept open two evenings in each week and on the minor holidays. In considering the budget for the ensuing year it was decided to continue this practice, as the additional expense, amounting to $704 is small compared to the benefit it offers. More books were consulted

during the past year and there were more visitors to the Library than in any previous year in the history of the College.

Under the will of the late Dr. Henry Leffman, a trust fund of $60,000 was created, one-third of the income from which is to be paid annually to the Library for its general purposes. The Library Committee has engaged Dr. Emlen Wood to act as assistant to the Librarian.

The Alvarenga prize was awarded by the Committee to Dr. Edgar S. J. King, of the University of Melbourne, Australia, for his paper entitled "The Nature of the Stroma of the Ovary."

The Mütter Museum: The Museum has been increasingly used for teaching and study throughout the year. At the meeting of the American Medical Association, in Philadelphia, in June, the Museum had a remarkable exhibit of specimens bearing on the subject of arthritis, and in connection with the meeting on the "History of Syphilis," held under the auspices of the Section of Medical History, in the Hall of the College on November 9, the Mütter Museum's extensive display of specimens excited much favorable comment. Arrangements have been completed whereby the Philadelphia Roentgen-ray Society will deposit its collection of films on bone tumors in the Museum, where it will be available for study. Dr. Meyer Solis-Cohen has presented to the Museum a large collection of laryngological instruments which belonged to his father, Dr. J. Solis-Cohen, one of the most distinguished pioneers in laryngoscopy in the United States. Dr. Astley P. C. Ashhurst presented to the Museum the microscope used by his father, Dr. John Ashhurst. Specimens of scientific interest were presented by Dr. Judson Deland and by Dr. Jonathan Wainwright, of Scranton.

The Nurses Directory. Next May our Directory for Nurses will have completed fifty years of unflagging service to the people of Philadelphia. In recent years it has had to encounter serious competition. Most of the hospitals have registries of their own, upon which their staffs are apt to call. A body of nurses has established a directory of their own in the last few years. Nevertheless, on November 30, 1931, there were 973 names on our registry, divided as follows: trained women nurses, 686; certified nurses, 45; male nurses, 75; attendants, 167; total, 973. During the year the Directory has filled 2636 calls. I am afraid that the splendid work done by the Committee of the Directory for Nurses is not as generally nor as fully recognized as it should be. It is not merely a

supply agency, but looks most carefully into the character and capacity of its registrants. Its discipline is very strict and it probably has done and is doing more to preserve the standards of the nursing profession than any other single force in this city.

The Sections of the College. They are all in flourishing condition; at their meetings papers of the greatest interest and value are presented not only by Fellows of the College but frequently by invited guests. The only criticism I find to make of the present relations between the College and its Sections is that there is a lack of coöperation between the chairman of the Sections and the Committee on Scientific Business of the College. If they would more frequently consult with one another on the arrangements made for future meetings and know more of one another's plans I believe that it would work for good for both the College and the Sections.

Deaths. During the past year the College has had the misfortune to lose by death fifteen Fellows, a larger proportion than usual. Memoirs will be read of Dr. Francis X. Dercum, Charles K. Mills, Hobart A. Hare, Charles H. Vinton, John B. Deaver and J. Madison Taylor; but it is my sad duty to chronicle the deaths of the following:

Dr. Samuel W. Morton, elected a Fellow of the College in 1894, died on January 12, 1931. In his earlier years in the profession Dr. Morton was an instructor in physical diagnosis and conducted a quiz class on medicine at the University of Pennsylvania. He had a very large general practice and was much beloved by his patients.

Dr. William Duffield Robinson, who became a Fellow in 1903, died, January 24, 1931. Dr. Robinson was very popular with his professional colleagues and enjoyed a large practice.

Dr. Harvey Shoemaker was elected to our Fellowship in 1893. After serving as resident physician in the Pennsylvania Hospital, he was for a number of years physician to the out-patient department. For many years he was a member of the staff of the Lankenau Hospital. Dr. Shoemaker acquired a very large practice and was held in the greatest affection and esteem by his patients, as well as by the members of his profession.

Dr. W. M. L. Ziegler became a Fellow in 1887. He died on May 29, 1931. Dr. Ziegler was a man of studious tastes, who had not been active in practice for a number of years.

Dr. Harry C. Deaver was elected a Fellow in 1894. His death occurred on June 25, 1931. Dr. Deaver was for many years surgeon to the Episcopal Hospital and to the Children's Hospital of the Mary J. Drexel Home. He was also surgeon-in-chief to the Kensington Hospital for Women. A surgeon of great ability, Dr. Deaver was also a delightful companion and friend. In the early years of his professional life he had a large quiz class on anatomy at the University of Pennsylvania, and his old students still recall his ability as a teacher and his kindness toward his pupils.

Dr. L. Napoleon Boston became a Fellow in 1913. His death occurred on July 4, 1931. Dr. Boston was a devoted and enthusiastic teacher of medicine. He was professor of physical diagnosis in the Graduate School of Medicine of the University of Pennsylvania, and professor of the principles and practice of medicine and clinical medicine in the Woman's Medical College. Besides writing an excellent textbook on *Physical Diagnosis*, Dr. Boston contributed much to current medical literature.

Dr. Horace H. Jenks was elected to Fellowship in 1919. He died, July 1, 1931. After serving as resident physician at the Children's and the Pennsylvania Hospitals, Dr. Jenks devoted himself entirely to pediatrics, in which specialty he attained well-deserved recognition. Most of his work was done in the Children's and St. Christopher's Hospitals, but he also taught in the Graduate School of the University of Pennsylvania. He did good work as medical director of the Associated Medical Clinics, and was an active and useful member of the committee on the Directory for Nurses of this College of Physicians. Of modest and retiring disposition and handicapped by ill health, Dr. Jenks leaves a splendid record of hard work and accomplishments.

Dr. George I. MacLeod was elected a Fellow in 1901. His death occurred, July 16, 1931. Early in his professional career Dr. MacLeod met with a terrible accident which overshadowed his whole subsequent life. He had opened an office at Ardmore and become associated with the late Dr. George S. Gerhard, who then enjoyed the largest practice along the Main Line of the Pennsylvania Railroad. One evening Dr. MacLeod slipped in boarding a train and received injuries requiring the amputation of both feet. His life hence forth was a heroic struggle against this handicap and the suffering incident to it. When able to get about he resumed his work and for many years maintained a large practice and attended

to his duties as one of the staff of the Bryn Mawr Hospital. In recent years he had been compelled to retire from such strenuous work and had devoted himself to psychiatry. Dr. MacLeod was a man of much culture and great charm and had a large circle of devoted patients and friends. During the Spanish-American War he served in Battery A, and during the Great War, in which he was unable to serve because of his physical incapacity, he did an immense amount of war work at home.

Dr. William L. Taylor was elected a Fellow in 1886. His death occurred, August 7, 1931. A quiet, retiring man, Dr. Taylor was a loyal Fellow of this College and a frequent attendant at its meetings.

SYMPOSIUM

THE THERAPEUTIC USE AND ABUSE OF FLUIDS*

Introduction. Purpose of the Symposium

By J. HAROLD AUSTIN, M.D.

It is a matter of profound regret that at this symposium, in which Dr. Hare was so interested, which he was largely responsible for initiating and concerning which he would have given us a clarifying point of view, we cannot have him with us owing to his illness, which we all trust will be a brief one.

I notice the title on the program is given as the "Use and Abuse of Fluids." In the original plan for this symposium the title read the "Use and Abuse of Fluids Intravenously," and this better indicates the field we are proposing to consider. There are certain forms of specific intravenous therapy which we do not propose to mention. No mention will be made of specific sera or antitoxins. No mention will be made either of intravenous drug therapy. There remains, when these large and important topics are excluded, two major therapeutic fields in which the intravenous method of administration is frequently utilized.

The first of these includes those therapeutic efforts directed toward supplying the body adequately with water, combating dehydration, the importance of which has been increasingly recognized of late, and by the administration of water promoting the excretory functions of the body. Important studies of recent years have thrown light on the relation that exists between the water and the salts in the body and have enabled us to recognize more clearly conditions demanding administration of water, conditions demanding administration of salt in water and conditions ameliorated by the administration of glucose in water.

The obvious channel of administering such fluids is in very many instances the mouth. Some part of what we propose to

* Read, January 7, 1931.

present is applicable to the principles that should guide us in administering such fluids by mouth. However, chiefly, we shall limit our discussion to the much more difficult problem of those instances where such fluids must, for one reason or another, be administered in large part parenterally and especially intravenously. It is in these therapeutic efforts that especial dangers are to be found, and it is these which especially justify thoughtful consideration and discussion.

The second therapeutic field in which intravenous channels of administration are frequently employed, and which we propose to discuss in this symposium, is that of nonspecific protein therapy. Dr. Kern will discuss certain aspects of nonspecific protein therapy in medical practice, certain of its dangers and some of its possibilities. Dr. Rademaker will deal with nonspecific protein reactions in surgical experience and with a recent application of nonspecific protein therapy that promises to be of value in both surgery and medicine.

That intravenous therapy is a method of treatment capable of doing the most serious damage must be kept constantly before us. One of the most conspicuous teachings derived from experimental physiology is the fact that the normal body, to a truly amazing degree, maintains an approximate constancy of composition in the blood serum, seeming thus to guard certain vital tissues from irreparable injury. To pass through the defences of the body and directly alter the serum composition is a procedure that is to be adopted only following the most careful consideration of all the facts that can be gathered concerning the effect of such a procedure, not only in the normal animal, but also, and this I think is of first importance, in an organism presenting the particular pathological limitations exhibited by the patient in hand. For this reason results of a beneficial character in one patient must be used with the utmost caution in considering the therapy of another patient whose pathologic condition is necessarily somewhat different.

In the discussion by the clinicians, medical and surgical, which will close the symposium, some of these safeguards and cautions which we must maintain in the practice of intravenous therapy will, I think, be emphasized.

INTRAVENOUS THERAPY IN DEHYDRATION AND TOXEMIA*

By J. HAROLD AUSTIN, M.D.

AND

I. S. RAVDIN, M.D.

Water is a necessary component of all living cells. In the higher forms of life the cells are bathed in a fluid medium which under normal conditions is remarkably constant in its organic and inorganic constituents. Variations in the chemical constitution, including water content, of the fluid bathing the cells vitally affects cell activity. The development of microchemical methods has extended greatly our knowledge of blood composition and its changes under various conditions, but as yet our knowledge of changes occurring in the tissues is most scanty.

When a patient has a much greater output than intake of fluid the tissue fluids and blood tend to become concentrated. Fluid continues to be lost through the kidneys, the skin and the lungs, although the amount is diminished. In an attempt to restore a normal blood concentration the water stores of the body are drained. The most readily available water stores are found in the skin, subcutaneous tissues and perhaps liver and muscles, and these are called upon first. Later the interstitial tissues lose water, while certain of the visceral organs are the last to give up their water stores. Associated with the loss of water which occurs in dehydration there is also a loss of inorganic constituents.

The immediate result in the patient is the same whether the dehydration is caused by decreased water intake or increased fluid loss. However, dehydration from lack of fluid intake can be repaired merely by the administration of water. Dehydration from fluid loss, such as occurs after vomiting, from severe diarrhea or following a duodenal fistula is invariably associated with the loss of salt, and repair demands the restoration not only of water but of salt also.

* Read January 7, 1931.

This differentiation of the types of dehydration has not received the emphasis which it requires for satisfactory therapeusis.

Animals and patients suffering from dehydration show evidences of thirst, the skin becomes dry, the urine concentrated and scanty, and the body temperature rises. A rapid loss of weight and strength follows. The viscosity of the blood increases, as the proportion of plasma to cells decreases; the hemoglobin rises as does the plasma protein.

The problem of dehydration concerns the clinician usually, when there exists an inability to swallow fluids; in persistent vomiting, or in severe diarrhea; when large amounts of fluid are lost through a discharging fistula, or when large amounts of fluid are lost from denuded surfaces, as occurs in extensive superficial burns.

It has become increasingly popular to administer fluids by the intravenous method. There are several reasons for this: first, the assurance that the fluid enters the blood stream; second, the ease and rapidity with which large amounts can be introduced in a relatively short period, and third, increasing evidence which makes it highly likely that glucose is not readily absorbed when given into the large bowel. Were it not for the frequency with which reactions follow the intravenous injections of saline or glucose there can be but little doubt that this method would have been even more widely used than it is. Even though post-injection reactions have in a measure limited the extent of intravenous therapy, we do not doubt that it is frequently used when safer routes are available. It is chiefly for this reason that we desire to review the indications for the intravenous use of glucose and saline for which a rational basis has been worked out in conditions associated with dehydration and toxemia.

Experimental studies have demonstrated that intravenous injections call forth activities on the part of the tissues which, with almost incredible rapidity, eliminate the major part of the changes in blood composition induced by the injection, and that the individual tissues participate in different degree and manner in this activity.

In the clinical use of intravenous therapy we would most like to know the changes that occur in the individual tissues secondary to changes induced in the blood. These appear to differ with differences in renal or hepatic function, the presence or absence of fever, the recent history of the organism with respect to water and salt balance

and the influence of drugs. Data concerning these changes are still scanty and generalizations are premature. We still have much to learn concerning these tissue reactions to intravenous injections.

Intravenous therapy with water, salt or glucose in hypotonic, isotonic or hypertonic solution should be adopted with assurance only in the treatment of the particular pathological conditions for which their value has been established and in the dosage and concentrations that have been shown to be helpful. To attempt to predict on general principles the effect of such therapy in other more or less related pathological conditions in which the effects have not actually been investigated is to court disaster. Each extension of the field of this therapy should be considered as a hazardous experiment to be conducted with extreme caution under conditions that permit complete study of the resulting effects.

The following carefully studied conditions in which such therapy has demonstrated its usefulness can be named.

In the dehydration of children associated with persistent vomiting and acidosis, in the pernicious vomiting of pregnancy, in high intestinal obstruction and in acute nephritis of children a carefully studied and successful therapy in the hands of Gamble, of Peters, of Hartman and others has been the combination of physiological saline solution given not intravenously but subcutaneously or intraperitoneally, or after vomiting has ceased, orally, in amounts up to a liter per day and at the same time the intravenous administration of glucose in distilled water, from 250 cc. of a 20 per cent solution to 1300 cc. of a 5 per cent solution per day. In these conditions, if there be definite nephritis with impaired renal function, the administration of saline intravenously has not only not been shown to be safe but is probably definitely dangerous. One apparent exception exists in a single carefully studied case of mercuric chloride poisoning with extreme oliguria, reported by Hayman and Priestly. In this patient striking and prompt improvement resulted from the administration for five days of from 1500 to 2800 cc. per day of 0.9 per cent sodium chloride solution intravenously, together with 3 to 4 liters of water per day by mouth. This result in bichloride poisoning, in which the renal picture is very different from that present in the more common types of nephritis, must not be taken as justifying the use of saline intravenously in other forms of renal disease.

Hayden and Orr, Whipple and others have successfully used

physiological or a slightly hypertonic saline solution intravenously to diminish the dehydration and the depletion of chlorides associated with high intestinal obstruction, under conditions where hypodermoclysis is not sufficiently rapid in its effects. Davidson and others have used physiological saline solution intravenously in the dehydration and chloride depletion of extensive superficial burns where hypodermoclysis is often impossible.

In diabetic acidosis with dehydration, Hartman has successfully used Ringer's solution containing 2 to 5 per cent of sodium bicarbonate intravenously in conjunction with glucose and insulin therapy. Peters, in similar cases, has given physiological saline subcutaneously and only the glucose and insulin intravenously.

For ten years surgeons have been preparing jaundiced patients for operation by administering glucose intravenously in amounts varying from 500 to 2000 cc. daily and in concentrations from 5 to 15 per cent, with or without physiological saline, for three to five days previous to operation, with a definite reduction in the operative morbidity and mortality. Mautner has shown that dehydration seriously impairs the ability of the liver to store glycogen. In these cases, therefore, we feel that the more dilute solutions (5 to 10 per cent) will give more satisfactory results than will the more concentrated glucose solutions. Furthermore, since the damaged liver is unable to take care of the injected glucose as rapidly as is the normal liver, the solution should be given very slowly. The possibility of obtaining equally satisfactory results from the administration of glucose by mouth has not received the attention which it merits. The normal patient will take care of about 0.8 to 1 gram of glucose per kilogram of body weight per hour without spilling over in the urine. Patients with an obstruction of the common duct will rarely take more than 0.3 to 0.4 gram per kilogram of body weight per hour. Thus, if the injected glucose is to be utilized it should be given very slowly. Glucose is just as important after such operations as before them.

If there are no contraindications to its administration orally this route should be used. If this is impossible the intravenous route may be used or the amount to be given may be divided between both routes.

In the vomiting associated with peritonitis and adynamic ileus the blood composition is practically the same as that seen in high intestinal obstruction. In these cases the subcutaneous or intra-

venous administration of physiological saline solution with the occasional administration of glucose has effectively counteracted dehydration and the ketonuria which is so often associated with this condition, especially in children. Although many of these patients have an actual alkalosis, there is frequently an associated ketonuria. The presence of ketone bodies in the urine has incorrectly led many surgeons to suppose they were dealing with an associated acidosis. The incorrect intravenous administration of sodium bicarbonate solutions to these cases is reprehensible.

As Drake, Marsh and Gamble have recently pointed out, it is often much more important to correct dehydration than it is to correct reaction; but, fortunately, in alkalosis with dehydration the treatment of dehydration is also the treatment for alkalosis – the administration of solutions of sodium chloride and of glucose.

In duodenal fistula, where large amounts of salts are lost, there occurs a marked deficit of salts as well as rapidly increasing dehydration. In these cases the administration of large amounts of physiological saline solution intravenously has been the means of saving a number of lives. The oral administration of fluids is useless since the injected fluid merely leaks from the duodenal fistula and sufficient fluids cannot be administered by other routes over a long period to take care of the fluid loss.

If fluids are administered intravenously certain important facts must be kept in mind. Not only must the ingredients be the purest that can be used, and the solution sterile, but if reactions are to be avoided the solution must be free from products of bacterial growth at the time of sterilization. To meet this requirement, glassware must be chemically cleaned just before use. Ordinary distilled water must be redistilled in such a way as to prevent transfer of toxic products of bacterial growth and the redistilled water used promptly for the preparation of the solution must be immediately sterilized.

We are using at present distilled water redistilled in glass with a trap filled with glass wool in the circuit, as described by Rademaker. This type of distillation prevents any toxic products from getting into the final solution. We are having installed a Barnstead still specially equipped for this purpose.

We are convinced that when fluids are to be given intravenously the slow, constant, intravenous drip is more satisfactory than the rapid introduction of large amounts in a relatively short period.

Reactions are much less frequent when fluids are given slowly. The reactions after intravenous infusions are probably largely due to two causes: as Seibert and Rademaker have pointed out, these may occur from the toxic products of a group of river bacteria which frequently contaminate the water and which can be avoided only by a specially chosen technique in preparing the solution. They probably, also, occur from the rapid introduction of large amounts of fluid, which strains the regulating mechanisms of the body.

Swale Vincent has demonstrated that the transitory fall in blood pressure which may accompany the intravenous injection of saline is more rapid the greater the rapidity of the injection, and we have confirmed these findings.

When the infusion is given slowly the circulation is not suddenly taxed by a large amount of fluid rapidly injected and the tissues are not called upon rapidly to regulate the electrolyte and water balance. With the slow-drop method, if intravenous infusion is indicated, it is the experience in the Department of Research Surgery that kidney output can be more adequately sustained, that the proper relationship between intake and output can be maintained and these without repeated puncture of an already severely taxed patient.

At this point we wish to emphasize the importance of wisely choosing the dosage of fluid administered. It is possible to give more fluid, especially when administered parentally, than is of benefit to the system. It is possible to induce dilution of the serum proteins and one may in such an event see clear evidence of detriment to the patient. This change is probably greater when saline is being injected. The aim of the therapy should be to restore the fluid lost and to provide a further intake equal to the daily loss through all channels, but to avoid exceeding this amount. For this reason it has been our practice to control our therapy by careful measurement of output, exclusive of that through the skin and lungs.

[Doctors Austin and Ravdin then cited several cases which demonstrated the value of intravenous therapy when carefully selected.]

Glucose was used in each of these cases, even when its need for combating ketonuria no longer existed. It served two purposes: first, in one instance it permitted the introduction of additional water, promoting diuresis, and it provided food, thus sparing to an extent the patient's tissues. The patients were unable to take fluids by mouth and we have no assurance that glucose is absorbed

from the rectum or large bowel. The fluid was given by a method of slow, intravenous drip, the rapidity of the drip being regulated by the amount which we desired the patient to have.

In summary we may state that, in combating dehydration from the loss of fluids by vomiting, diarrhea, exudation or excretion through a fistula; by whatever route of administration, be it oral, subcutaneous or intravenous, saline rather than water is required.

The rapid intravenous administration of saline is associated with dangers, notably in the presence of impaired renal function, and also when, as a result of the toxemia, there is a failing circulation.

The slow, intravenous administration of saline by the drop method, in the experience of our surgical clinic, combines a high degree of safety with marked efficiency and provides in cases where sufficient fluids cannot be introduced by other routes or where there exist definite contraindications to such other routes, the method of choice in combating dehydration.

Glucose intravenously without saline has been used effectively in combating certain forms of nephritis and ketosis; it is the method of choice for introducing water parenterally when water alone, free from salt, is desired and it possesses at the same time food value.

In intravenous saline or glucose therapy not only is the choice of fluid and its amount of importance but of great importance is the preparation of the solutions and the rate of injection.

NONSPECIFIC PROTEIN THERAPY*

Some Cautions as to Its Use

By RICHARD A. KERN, M.D.

(From the Medical Division, Hospital of the University of Pennsylvania)

I. Introduction. While the earlier work on the immunology and treatment of infections was based on a strict specificity, it soon became apparent that certain therapeutic results were undoubtedly of a nonspecific nature. Specific vaccines administered for the treatment of certain infections were unexpectedly found to exert at times a beneficial effect on unrelated but accidentally co-existent infections. This was noted in prophylaxis as well. For example, Sir Almroth Wright, when he first gave pneumococcus vaccine to the workers in the Premier Mine, in the Transvaal, noted not only a reduction of 85 per cent in pneumonia mortality, but also a 50 per cent reduction in the mortality from other diseases. Of similar significance was the common clinical experience that an intercurrent acute febrile infectious illness had at times a strikingly beneficial effect upon a chronic infection from which the patient was also suffering. The investigation of these phenomena and their practical application led to the development of a new and important addition to the treatment of infections: nonspecific or protein therapy.

II. The Nonspecific Reaction. The parenteral introduction into the body, usually by intravenous or intramuscular injection, of a great variety of substances, usually protein in nature but not necessarily so, is followed by a reaction varying from a mere stimulation of leukocytes to mild febrile reactions and to extreme shock pictures with profound vasomotor paralysis and even death. In its typical and often most desirable form the reaction shows first a chill, then fever, a pulse rise, a fall in blood-pressure (due in part to peripheral dilatation of bloodvessels), sweating, occasionally

* Read January 7, 1931.

nausea and vomiting; a primary fall, then a rise of leukocytes. There are profound changes in cellular activity including altered permeability of cell walls, a primary depression and then marked stimulation of function. At the site of an existing infection, occurs the so-called *focal reaction* with at first an increase of the local disease process, and then, in favorably reacting cases, a regression. Subjectively, the patient experiences at first intense discomfort, then a striking euphoria. Clinical improvement of infection is probably the result of greatly increased cellular function, including the production of specific immune substances—a sudden and massive mobilization, as it were, of the whole body's defense mechanism.

III. Its Biphasic Nature. It is essential to recognize the biphasic nature of the reaction: invariably an initial depression, which if too severe may harm or even kill; then the desired stimulation which up to a certain limit depends in degree upon the severity of the preceding depression, and which is further conditioned by the ability of the body to respond: an organism fatigued to the point of exhaustion is no longer capable of stimulation.

IV. Factors Conditioning the Reaction. The type and degree of reaction depend upon a number of factors. First of all, is the *nature of the substance used* to produce the reaction. Literally scores of agents have been employed, and no two of them produce exactly the same effect. Then obviously to be considered are the *mode of administration* (whether subcutaneous, intramuscular or intravenous), *the dosage*, and the *number of previous injections*. The *type of infection under treatment* is of course important. In acute infections, like pneumonia and typhoid fever, the reaction is less constant in its manifestations, and the depression phase of a shock reaction may be far too hazardous to risk. On the other hand, some of the most brilliant successes of the method have been obtained in acute rheumatic fever.

In chronic infections the reactions are more constant and seen to better advantage. There is one outstanding exception, tuberculosis, in which this treatment is never helpful but always harmful. Above all, the *condition of the patient* himself is a factor in determining the reaction.

V. Certain Cautions to be Observed. With these facts in mind, and in view of the widespread and often haphazard use of nonspecific protein therapy, it is little wonder that many and at times grievous errors have been committed. One may, therefore,

be permitted to stress certain cautions to be observed by those who plan to make use of this therapeutic measure.

(*a*) We must be thoroughly familiar with the substances used their preparation, dosage and the type of reaction produced. For instance, in the case of killed typhoid bacilli, there may be a wide difference in severity of reaction produced by equal amounts of different strains of the organism; one must learn by experience the proper dosage of a certain typhoid bacterial suspension, and then stick to that preparation. Again: the sharp severe reaction which usually follows intravenous typhoid bacterin injection may be too severe for a given patient and we must turn to the milder effects that are produced by intramuscular milk or casein injections.

(*b*) It is a fortunate thing that man is not as easily sensitized to foreign proteins as is the guinea-pig, or else there would be far more disasters to record. But the fact remains, that man can at times be so sensitized. We should, therefore, avoid the use of substances which are known to act as sensitizing antigens, notably horse serum, egg albumen and perhaps also milk and casein. Yet ophthalmologists have been particularly partial to horse serum in the treatment of certain eye conditions such as retinitis and keratitis.

(*c*) We must remember, too, that an appreciable number of individuals are naturally hypersensitive to a wide range of substances, and that in such persons a corresponding injection may prove fatal. It should be an invariable rule, therefore, to inquire into the history for personal or familial manifestations of allergy. This does not necessarily mean that in such persons nonspecific therapy should never be employed. We might consider the treatment of paresis by malaria inoculation as one of the newest forms of nonspecific therapy. Yet in 1818 Dr. Nathaniel Chapman, in his lectures to medical students at the University of Pennsylvania, stated that it was a common practice of European physicians to send asthmatic patients to the Mediterranean littoral for the benefit that often followed an attack of malarial fever. Today, nonspecific therapy has a definite, though minor, place in combating the frequently attendant and at times causal respiratory tract infection in bronchial asthma.

(*d*) When a first-shock reaction has been followed by slight or moderate improvement of the disease under treatment, the natural thought is to repeat the procedure one or more times. Yet it is true that subsequent injections of even greatly increased doses of

most agents, especially if too closely spaced, give progressively lesser reactions and correspondingly less good results. It is a fatigue phenomenon that cannot be ignored.

(*e*) We must carefully consider the recognized contraindications. These have primarily to do with the condition of the patient. Hypersensitiveness has been referred to. Cardiovascular disease, (including severe valvular lesions and myocardial weakness, high blood-pressure and marked arteriosclerosis) is an absolute contraindication. So also are pregnancy and diabetes. In chronic alcoholics a shock reaction may precipitate a fatal delirium tremens.

A patient who is already excessively worn out by his illness can hardly be expected to respond favorably to so harsh a stimulus. In short, we should never use the measure unless the patient is a good clinical risk.

(*f*) Finally, *nonspecific* therapy should in no way replace *specific* treatment. Specific measures are always to be employed first, with nonspecific therapy as an avilable but adjuvant measure. Yet frequently enough we meet infections for which no specific treatment is known and in such cases protein-shock therapy has at times shown its greatest usefulness.

VI. Conclusion. I can best conclude by quoting Peterson[1] when he says: "Nonspecific treatment requires judgment, careful attention and bedside study on the part of the physician perhaps in greater measure than any other therapeutic procedure. It should never be a routine; to be useful it must be an individualized therapy, with dosage and preparation and time of application varied to the disease, its intensity, its duration and the resistance of the patient."

REFERENCE

1. Peterson, William F.: Protein Therapy and Nonspecific Resistance, New York, Macmillan Company, 1922.

NONSPECIFIC PROTEIN THERAPY IN SURGICAL CONDITIONS*

By LEE RADEMAKER, M.D.
By Invitation

The application of foreign protein therapy to surgical conditions was first described and advocated by Brown in 1926. While studying heat elimination and skin temperatures of patients suffering with vascular disturbances of the extremities and chronic arthritis, it was found that there was a great increase of heat elimination during the febrile stage of the reaction. This is logically explained as the compensatory mechanism of the body to decrease the artificially increased body temperature by increasing heat elimination where body surface is greatest and vessels are nearest the surface. During this period of increased heat of the extremities the symptoms due to poor blood supply were found markedly diminished, pain was relieved, cyanosis disappeared and the patient showed improvement which persisted for a time after the rise of temperature had subsided. In addition to this, Brown found that the increase of surface temperature and heat elimination was exactly as great as that which could be obtained in the same patient by sympathetic ganglionectomy as practised by Adson. Studies of capillary flow by Tremont-Smith have substantiated Brown's findings and have shown that the improvement is definitely due to increase in bloodflow. Brown reported 15 cases of vascular disturbances of the extremities treated with intravenous typhoid vaccine, a large percentage of which were markedly improved. From this work has come the later application of foreign protein therapy in cases of vascular disturbance. Having noted that the effect of typhoid treatment on skin temperature was the same as the effect of ganglionectomy, this fact was applied for appraising the value of ganglionectomy in the individual patient. Adson and Brown use the results obtained after the intravenous injection of typhoid vaccine to obtain a vasomotor index. This is defined as the

* Read January 7, 1931.

ratio of the change in skin temperature minus the change in mouth temperature to the change in mouth temperature. When this ratio is larger than $1\frac{1}{2}$, good results, with good dilatation of vessels can be expected from ganglionectomy. When the index is below this figure ganglionectomy is of no value, because no appreciable amount of improvement may be expected.

This index naturally divides patients suffering with vascular disturbances into three groups: those with a good vasomotor index with mild or insignificant symptoms, those with a poor vasomotor index and those with a good vasomotor index with severe or moderate symptoms. In the latter group ganglionectomy is obviously the treatment of choice because of the permanent results. It is in the first two groups that foreign protein therapy finds its greatest value. In patients with mild symptoms protein therapy can be expected to allay all symptoms and check, at least temporarily, the progress of the disease until the growth of collateral circulation has a wide margin of safety over the obliterative process and gangrene is not likely to occur. In the second group, in which ganglionectomy is of no value, the inevitable result has always heretofore been gangrene with amputation and often reamputation, although the process may take years to reach this stage. With foreign protein therapy the growth of collateral circulation may often be kept ahead of the obliterative process and gangrene be avoided.

Allen and Smithwick reported 25 cases of vascular disturbances of the extremities treated with typhoid vaccines, all of which showed markedly good results when observed over periods of one to eighteen months. Their technique was to use 125,000,000 typhoid organisms intravenously as a start and increase or decrease as necessary, repeating once per week, with rest periods between series of 4 or 5 injections. They call attention to the possibility of thrombosis in arteriosclerotic patients during the prereaction period of shock, when blood-flow has been shown to be definitely slowed.

Since the time of Brown's report we have treated 15 cases in the University of Pennsylvania Hospital, most of whom were on the surgical service of Dr. Müller. In this number we have had 1 case of thrombosis in an arteriosclerotic patient with ulcers on a leg stump, the previous amputation having been for arteriosclerotic gangrene. In all others there has been improvement, sometimes marked and permanent, at other times only temporary. The technique has followed that of Allen and Smithwick, except that the

initial dose has been 50,000,000 organisms, which in course of treatment has been gradually increased until in some cases 400,000,000 were used. The pyrogenic elements of distilled water have also been used in these cases, but results have not on the whole been as good as with typhoid vaccine, although the untoward symptoms such as nausea, headache and bone pains have been entirely lacking when this solution was used. Eight of our patients had thrombo-angiitis obliterans, 2 had Raynaud's disease, 3 had chronic arthritis and 1 had scleroderma. It is especially in the first two groups that we expect this form of treatment to be of value and we are showing tonight the effects on 5 cases. The apparatus of Bazett and McGlone was used for determining skin temperatures.

Case I.—J. R., aged sixty-eight years, had rapidly progressing thrombo-angiitis obliterans in both legs, of two years' duration. The first typhoid treatment, combined with a left femoral periarterial sympathectomy, produced a good rise in skin temperatures and abated all symptoms. Most of this improvement was probably due to the typhoid vaccine, since the skin temperatures of both legs were equally elevated to almost normal. Subsequently, however, the process of obliteration continued, especially in the right leg. A section of very fragile, occluded femoral artery was removed on the right side when its fragility prevented periarterial sympathectomy as planned. Typhoid vaccine treatments were continued. Although this patient's vasomotor index was zero and only very little elevation of skin temperature could be shown, his symptoms were completely relieved for a few days at each treatment. No other measures, even large doses of morphine, were capable of producing this effect. Two months later ulcers had failed to heal on his right foot, although the left foot had healed and become painless and a Gritti-Stokes amputation of the right leg was done. The stump healed and the patient has remained free of symptoms for a year.

Case II.—M. F., aged thirty-three years, also has thrombo-angiitis obliterans, of seven years' standing. His left leg had been amputated beneath the knee, the right leg was blue and cold and ulcers were present on the heel and toes. A series of typhoid vaccine treatments healed the ulcers and relieved his pain. An ulcer on the stump improved but did not completely heal, his vasomotor index was also zero, and continued treatment has kept his right foot in excellent condition. The left stump has needed reamputation, because of chronic ulcers. At reamputation, however, the collateral vessels were found markedly dilated, and appeared ample to supply the part. Local mistreatment of the stump by the patient despite all advice was probably the cause of the ulcers not healing. Skin temperatures of the right leg are now practically normal.

Case III.—F. B., aged thirty-nine years, is probably the most remarkable. The patient had thrombo-angiitis obliterans for twelve years, resulting in multiple amputations of his right leg, the final amputation being just below the hip-joint. On admission his hands were cold and blue, and there was beginning gangrene of several fingers. Typhoid vaccine treatment has restored completely the skin temperature of his hands and of the right leg to normal. All symptoms have been relieved and although pulsations are absent in many of his large vessels, the collateral circulation has probably compensated for the obliteration of the larger vessels. This patient also had a zero vasomotor index, so that ramisection or ganglionectomy was of no value.

Case IV is also one of thrombo-angiitis obliterans, this time of a much milder character. J. W., aged fifty-four years, was first seen in July, 1929, complained of intermittent claudication and pains of walking of seven months' duration. This patient's skin temperatures after a series of treatments were not markedly subnormal and the vasomotor index was therefore rather small. His symptoms were moreover not severe enough to justify ganglionectomy at this time. Vaccine therapy has kept him comfortable, so that he is able to walk and work daily with but little trouble.

Case V is one of Raynaud's disease with a confusing superimposed picture of thrombo-angiitis obliterans. M. L., aged forty-three years, was admitted May, 1928, with partial gangrene of and blisters on five of his fingers. His hands were blue and cold and he was unable to work. After two active courses of treatment all symptoms disappeared and since that time he has had occasional treatments with entire and complete relief of symptoms. His skin temperatures are now normal and he is able to do all his work as tailor without difficulty. His fingers occasionally become slighty stiff in cold weather, but this is his only symptom. Pulsations are absent in all palpable arteries at both wrists.

Foreign protein therapy thus has a definite value in deciding the value of sympathetic ganglionectomy in patients suffering with vascular disturbances of the extremities. Caution must once more be urged because of the possibility of thrombosis in arteriosclerotic patients, since this danger is very real and by no means fancied. It also has a therapeutic value in the two groups of cases in which ganglionectomy is of no avail and for which as yet we have no other more satisfactory method of treatment.

DISCUSSION FROM A SURGICAL STANDPOINT*

By JOHN H. GIBBON, M.D.

A SURGEON should not become too facile in performance of any one operation because he is apt to see occasion for it when it does not exist. It is so much easier to learn the treatment of the disease than to learn the disease itself. Intravenous therapy has been a boon to surgery. Dehydration has been one of the very potent factors in our surgical mortality and we have learned now that it must be overcome before operation and it must be combated after operation. The administration of fluids by rectum, under the skin and by the vein has saved the lives of hundreds and thousands of patients that otherwise would have been lost. In the communications that have been presented to us tonight caution has been a prominent feature in all of them. We have been told why and when this method of treatment should not be employed. We have been told that the intravenous adminstration of fluids by the vein is to be employed when their use under the skin or by rectum cannot be employed or where it is not sufficient. We cannot give food under the skin and it is a question how much absorption of glucose occurs in the victims. I have been astounded by how long a time patients can be kept alive by the continuous intravenous glucose drip. We see the most dramatic results in intestinal obstruction and in upper intestinal fistula.

Dr. Raydin's case reminds me of a case I saw a year or two ago. The patient had been operated upon for a bad appendicitis and developed intestinal obstruction. Jejunostomy and ileostomy had been done, and when I saw this patient he was the picture of starvation. He had wasted away to a skeleton. His skin was dry and cracked and he was losing quarts of fluid. They were collecting the fluid from the jejunal tube and injecting into the ileal tube. The patient was getting practically no fluid. It was only when subcutaneous fluid and glucose was started that the patient began to get well.

* Read, January 7, 1931.

These agents are capable of great abuse; it is just like transfusion. There is no method of treatment that has not been improperly used and abused. Transfusions are given now before hemorrhage is controlled. Gastrojejunostomy, as you know, was so abused that it was done for the cure of epilepsy. So in considering these very valuable methods, we must remember that they have their limitations and they are very definite, that they have their contraindications which are very definite, that through improper use serious consequences in the form of reaction may follow. If we pay as much attention to the precautions laid down in these papers as to the use and also as to the preparation of the fluids we use, then I think we will have got a great deal from these papers; but I think it is very important to pay particular attention to that side of these presentations.

I was particularly interested in what was said regarding nonprotein therapy in certain arterial conditions. I had some difficulty in understanding the permanence or the duration of the results obtained by the combination of periarterial sympathectomy and the nonprotein therapy in the cases of Burger's disease, and I hope Dr. Rademaker will tell us when he comes to close the discussion.

DISCUSSION FROM A SURGICAL STANDPOINT*

By DAMON B. PFEIFFER, M.D.

At the risk of submitting you to the tedium of a one case report I think I can best add to this symposium by a personal experience. This concerns a man, aged forty-one years, admitted to the Presbyterian Hospital, September a year ago, under the care of Dr. Phillips. He was semistuporous, irrational and at intervals vomited profusely. He was thought to be uremic of nephritic origin, diagnosis being based on the peculiar pallor, nephritic facies, red blood cells, high blood urea nitrogen. His blood-pressure, however, was low, 108 over 68. He was treated by forced fluid and eliminative measures with very satisfactory results; he responded immediately and in the course of two or three weeks had regained his usual condition of health. It was obvious, however, that he had gastric retention and I was asked to see him for that reason. His gastric history was interesting. At the age of fourteen years he had an attack of whooping-cough following which he was troubled by digestive symptoms, and continued to be troubled up to the present time, age of forty-one years. In the early period there was pain associated with eating, but as time went on pain ceased to be a prominent feature and he had only distress after meals which was relieved by vomiting and the use of the stomach tube. A previous *x*-ray examination had lead to a diagnosis of visceroptosis. This was repeated at the Presbyterian Hospital and the roentgenologist verified ptosis and said there was deformity of the cap which indicated adhesions. In view of the fact that he was getting better so rapidly we thought we would not advise surgical intervention, but sent him home under a régime calculated to take care of his ptosis and nephritis and he continued to improve for four or five weeks and became very much better. He then began to vomit again and relapsed into an anemic state. He was admitted seven weeks later semistuporous, vomiting, irrational, and on this occasion the medical treatment which had proven efficacious on the previous admission failed. He grew steadily

* Read, January 7, 1931.

worse. When I saw him on the seventeenth day he seemed almost moribund, was restless, muscles were twitching, he was unconscious and still continued to vomit at intervals, though the stomach was washed out. His blood urea nitrogen at this time was 100. It was thought this might be a case not of uremia but of alkalosis. On taking his CO_2 and chlorides we found that hypothesis verified, chlorides 180, only about one-third normal and CO_2 98 volume per cent, about twice normal. We decided, therefore, to abandon the treatment of uremia as such and concentrated our efforts to restore the blood chemistry to normal. At this period the chlorides were 180, plasma 102, CO_2 about 98. He was given salt as energetically as it had been denied. We gave saline 1000 cc. of 10% glucose solution at a time, and calcium chloride. Within twenty-four hours he was conscious, passed several thousand cubic centimeters of urine and the blood chemistry had improved very markedly. His blood urea nitrogen dropped very little. CO_2 was down to 72. The next day he made still further gains. The next day he was still further improved. He then showed a curious phenomenon of gastric hypersecretion. Though nothing was being administered by mouth, that day 2800 cc. of biliary fluid was drawn from his stomach. By repeated efforts, however, he gained and, finally, at the end of a week his blood chemistry had returned to normal, with the exception of blood urea nitrogen which remained rather high. As it approached normal it was interesting to see the fluid from the stomach decrease. He went along on parenteral fluid for twelve days. During that time I do not believe a particle of fluid went from the stomach into the intestine. We had to siphon it from the stomach. I did a jejunostomy under local anesthesia and it was then possible to give fluid by the intestine. This was carried out for two weeks more. We would try out the stomach and found that nothing went through. Later on we found, by means of bismuth paste and *x*-ray studies, that he had an organic stenosis of the pylorus. Spinal anesthesia was selected although the blood-pressure was only 90; I felt it would be safer than general anesthesia. With the aid of ephedrin he went on the operating table with blood-pressure of 90 and came off with blood-pressure of 128. There was no difficulty about it whatever. He had the scar of an old ulcer of the pylorus. Posterior gastroenterostomy was done and the appendix was removed. I felt reassured on account of having the jejunostomy, because if gastroenterostomy

did not function successfully we could fall back on the jejunum. After the operation whatever was introduced into the jejunum returned into the stomach and this continued for several days. In three days, however, he started to retain what was given by mouth. He left the hospital in three weeks, gained 25 pounds in two months and is doing well now. This is an extreme case and I think the spirit of this meeting is to advocate more exact recognition of the indications for and methods of using fluid therapy.

Dr. L. A. Rademaker: In reply to Dr. Gibbon's question, these cases have been treated over a period of two and some of them two and a half years. The permanence of the effect depends almost entirely upon the obliterative process and its rapidity of progression. The periarterial effect in the one case has been added to by the use of typhoid vaccine and has remained perfectly normal and free from symptoms for a period of eighteen months since the operation.

SYMPATHETIC ACTIVITY IN CERTAIN DISEASES, ESPECIALLY THOSE OF THE PERIPHERAL CIRCULATION*

THE MARY SCOTT NEWBOLD LECTURE
LECTURE XXV

BY W. J. MERLE SCOTT, M.D.
AND
JOHN J. MORTON, M.D.

(From the Department of Surgery, The University of Rochester School of Medicine and Dentistry, Rochester, N. Y.)

FUNDAMENTAL facts concerning the anatomy and physiology of the sympathetic nervous system lay dormant for many decades. At the very end of the nineteenth century several attempts were made to influence the course of epilepsy, exophthalmic goiter and glaucoma by interrupting or excising parts of the cervical sympathetic chain. These efforts had been almost forgotten, due to the poor results achieved, when in 1916 Jonnesco attracted the attention of the medical world by performing cervical sympathectomy for angina pectoris. The interest in the removal of the sympathetic influence was greatly quickened by Leriche's work on periarterial sympathectomy, which he first employed in 1917 in the treatment of causalgia and in subsequent years for other conditions attributed to vasomotor disturbances. The eradication of visceral pain by interrupting sympathetic impulses, either temporarily as a diagnostic measure or permanently at operation, further attracted attention in recent years to this system. This interest, however, has been very greatly stimulated since the work of Hunter and Royle in 1924. This episode illustrates beautifully the value of painstaking clinical observation. There is still today, after a great deal of clinical and experimental work, much doubt concerning the major point of their investigation, namely, the influence of

* Read, February 4, 1931.

sympathetic impulses on muscular spasticity. But two of their incidental observations have opened up important lines of attack in fields quite unrelated to that of their primary objective. They noted that the obstinate constipation shown by several of their spastic paralytic patients was overcome after lumbar sympathetic ramisection and also that the foot on the operated side became much warmer. Operations to interrupt the lumbar sympathetic nerves were then undertaken as empirical procedures by various surgeons in thromboangiitis obliterans, Raynaud's disease and Hirschsprung's disease. Dr. Elliott C. Cutler and one of us performed lumbar sympathetic ganglionectomy on 2 cases of thromboangiitis obliterans a few weeks after the visit of Hunter and Royle to this country in 1924. These patients were selected as their symptoms seemed to afford a good basis on which to judge of improvement. Both had severe, almost unbearable, nocturnal pain with the usual manifestations of greatly impaired circulation in the foot. In 1 case a large and slowly progressing ulcer on the dorsum of the foot was evidence of serious circulatory deficiency. The latter patient showed no benefit from sympathectomy and required amputation soon afterward. The other patient showed definite clinical improvement; his paroxysms of excruciating pain were almost entirely controlled and a gangrenous area on one toe slowly healed. The patient was able to use his foot for several months, but returned within a year with further gangrene for which amputation was done. From results of sympathetic interruption reported in the literature it soon became apparent that some benefit was being achieved in certain cases, while in others the operation failed to materially affect the disease. In fact, this pioneer work has indicated a number of pathological fields where sympathetic activity may play a leading rôle in the production of harmful effects. From the first, however, an important obstacle to the clinical exploitation of these possibilities has been the lack of accurate criteria by which to demonstrate and to measure the element of sympathetic activity in the clinical syndromes.

In 1929 this need presented itself to us in deciding upon the treatment of a patient with a typical example of Hirschsprung's disease. At that time the results of interrupting the sympathetic innervation of the large intestine for this condition were recorded in the literature in only 7 cases. In 5 of these the immediate result was distinctly gratifying, although the interval of postopera-

tive observation was too short to warrant final conclusions. If sympathectomy would cure this patient we surely wanted to carry it out. On the other hand, if it would not benefit this particular child we did not want to submit him to the operation unnecessarily. We reasoned that if the abnormality was dependent upon activity of the lumbar sympathetic nerves and would be benefited by their removal, then temporary improvement in the motility of the bowel should be demonstrated after paralyzing the lumbar efferent sympathetic fibers from the cord. Consequently we determined the motor function of the bowel by a barium enema before and after the induction of spinal anesthesia.* The effect of this procedure was so striking that we were convinced that the patient would be greatly benefited by permanent interruption of the lumbar sympathetic outflow. After bilateral lumbar sympathetic ganglionectomy the patient began having regular bowel movements and became capable of completely emptying the contents of the left colon, as demonstrated by a motor test with a barium enema. We have applied this method with similar convincing results in another case of Hirschsprung's disease. It also has shown its value in 2 cases of obstinate constipation not of the Hirschsprung type. These latter patients did not show the rapid emptying of the distended bowel after spinal anesthesia, as did the former. We concluded, therefore, that the constipation arose on some basis other than sympathetic activity and that sympathectomy would not relieve their obstipation. In order to demonstrate further the effect of spinal anesthesia on the motor mechanism of the large bowel, this was studied in the cat. Under the conditions of the experiment, with the animals under light ether anesthesia and the straight gut exposed and kept moist with a fine saline spray, it showed no effective peristaltic or expulsive action, even when fully distended with fluid. Shortly after the introduction of procaine intraspinally, vigorous coördinated peristalsis appeared, first in the cecal region followed in a short time by powerful expulsive contractions, resulting, when the bowel was distended, in the emptying of its contents. From this clinical and experimental work we concluded that spinal anesthesia was a satisfactory method for testing the presence of sympathetic inhibition acting on the large bowel. It also seemed reasonable to expect that this method would prove

* Roentgenograms of this procedure were shown. They will be found reproduced in one of our previous papers (1).

equally effective in connection with other systems under the influence of these nerves. We turned our attention, therefore, to its application in diseases of the peripheral vascular system.

"In those cases of gangrene in which no obstruction has been found after death in the vessels of the dead parts it is extremely probable that a long, persistent spasm of the bloodvessels has existed." Thus Brown-Sequard,[2] in a lecture before the Royal College of Surgeons of England, in May, 1858, called attention to the clinical importance of vascular spasm and its differentiation from occlusion. In the early part of the same decade Claude Bernard had demonstrated the vasoconstrictor fibers in the cervical sympathetic chain. The physiological action and anatomical pathways of the sympathetic vasoconstrictors are well known. This information, however, had not been applied to the development of a practical method for differentiating the elements of spasm and occlusion and for determining their relative participation in any individual case of peripheral vascular disease. As will appear later, this distinction is of great significance in the common forms of vascular disease, not merely in the unusual instances such as those alluded to by Brown-Sequard and those described by Raynaud, in 1862, in his classical monograph on local asphyxia and symmetrical gangrene of the extremities. French investigators,[3] by the use of the oscillometer before and after immersing the extremity in a hot water-bath, had attempted this distinction. However, this method gave no index to the bloodflow through the finer vessels of the collateral and terminal circulation. It is inadequate for our purposes. The hyperthermia following the parenteral administration of a foreign protein had long ago been advised as a method of treatment in many conditions, among them the peripheral vascular diseases. Brown[4] applied this reaction to the diagnosis of these peripheral vascular diseases, deriving a vasomotor index from a comparison of the increase in surface temperature of the foot with the increase in body temperature. This method really offered the first differentiation of spasm and occlusion in the involved area. We have used it several times, but it is disagreeable to the patients, inconvenient to the physician and in some reported cases[5] has resulted in serious complications. Consequently we applied ourselves to the development of a simpler procedure to measure the elements of spasm and occlusion in the extremity. It was an obvious step to carry out

the same procedure in vascular disease that had been so satisfactory as a test for sympathetic activity in Hirschsprung's disease. We, therefore, studied the effect of spinal anesthesia upon the circulation through the extremities in individuals with normal vascular systems as a basis on which to interpret the response in pathological cases.

Let us interpose here a few remarks about the method chosen to measure the vasomotor changes in the extremities. Three such are available and have been used in studying the peripheral circu-

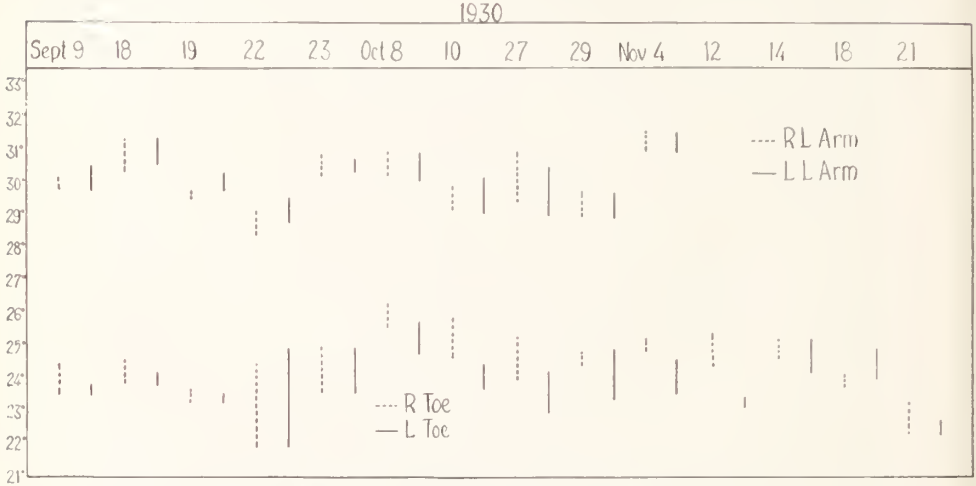

FIG. 1.—Skin temperature variations of a normal subject (Miss O. E.) in a room at a constant temperature (20° C.) (68° F.). Each line represents the maximum and minimum readings over a period of one-half hour. The area of skin was exposed to the standard conditions for fifteen minutes each time before temperature readings were started.

lation in the laboratory and in the clinic. These are: (1) Plethysmographic records of volume changes; (2) the rate of heat dissipation from the part, usually by a water calorimeter; (3) surface temperature determinations. The plethysmographic and calorimetric methods, though sensitive indices of the rate of bloodflow are not applicable to comparative determinations in various areas such as the different toes, or the toe as compared with the sole, etc., data which, as will appear later, is of importance to us. They are available in clinical use only when applied to larger parts, such as the whole foot or lower leg. The surface temperature, however,

can be easily and quickly determined for any number of points. At a given room temperature it is an accurate index of the blood-flow through the involved area, if this does not closely overlie contracting muscles. Surface temperatures in normal individuals vary greatly. (Fig. 1.) What, then, is their significance and how are they of use to us in studying this problem? The actual reading at which the surface temperature starts is of little significance. It is an index of the arteriolar circulation which is undergoing many physiological changes during the day under normal conditions, as is well known. Comparative temperatures are sometimes of importance, especially when a difference of at least 1° C. is shown in

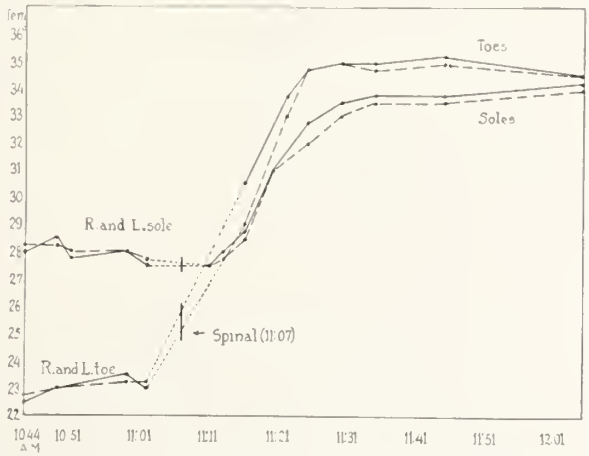

FIG. 2.—(Case 32599.) Normal response to spinal anesthesia in a case in which herniotomy was performed on the right side (normal bloodvessels). Temperature of toes and soles rise sharply and come to the same level.

symmetrical areas. The chief value of surface temperature determinations, however, consists in following their response to certain procedures which normally affect the vasomotor activity in the extremity. Thus if vasoconstriction is well marked, as it normally is, or can be induced in the distal part of the lower extremity, the surface temperature should show a striking increase on paralyzing the sympathetic vasoconstrictor fibers.

We determined the surface temperatures in a group of individuals with apparently normal vascular systems before and after the induction of spinal anesthesia for various types of operations. (Fig. 2.) With the exception of a small group, which I will presently

discuss, all of these patients showed a sharp increase in the surface temperatures of the feet upon the establishment of complete anesthesia in that area. After a very few observations it was evident that this furnished a satisfactory basis for demonstrating the presence of vasoconstriction. We later learned that simultaneously in at least two other clinics this same procedure was being developed as a qualitative test for vasoconstriction.[6,7] After assembling a group of 22 such curves in normal individuals a careful study of them yielded further important information. Those curves showing a sharp rise in surface temperatures regularly reached a level at

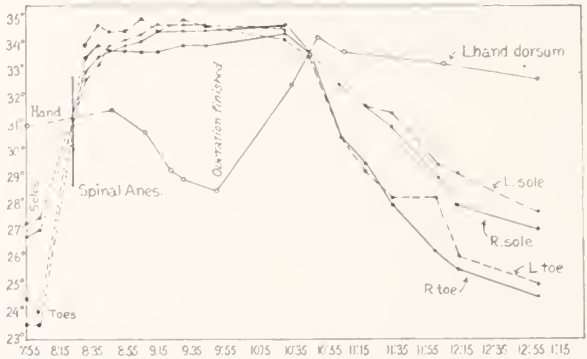

FIG. 3.—(W. S., Case 34172.) A normal vasodilatation level, reached by the toes and soles following spinal anesthesia in a case in which herniotomy was performed, is maintained as long as the complete anesthesia lasts (about two hours). As sensation and motion return to the extremities the temperatures of the toes and of the soles fall approximately to the point from which they started (reappearance of vasoconstrictor gradient). The hand, not being anesthetized, shows a fall in surface temperature during the operation (psychic and mechanical influences). The room temperature was 24.9° C.

which they remained for a considerable time, then gradually declined to approximately the original point. (Fig. 3.) Furthermore, the level which the colder part of the extremity (usually the toes) attained was approximately the same on both sides and also the same one which other parts of the extremity came to. Upon comparing this with the original temperature of the warmest part of the exposed body, again it was approximately the same. Upon studying these results mathematically, we were surprised to find in these 22 cases that the height of the temperature attained by the surface of the great toe always fell within 1.5° C. of the mean. In other words, even without correction for room temperature, which

varied considerably in this first series, the skin in these normal extremities became approximately uniformly warmer not only throughout the extremity but on comparison of one individual with another. We named this level attained the *normal vasodilatation level*,[8] as it seemed to represent the circulation entirely freed from vasoconstrictor impulses. Three cases were found which did not respond by a sharp rise in surface temperatures after satisfactory spinal anesthesia. In these patients (Fig. 4), though the peripheral circulation did not appear grossly abnormal, the skin temperature of the toes and soles started at a very high level, in fact, nearly at the vasodilatation level, and either remained about the same or

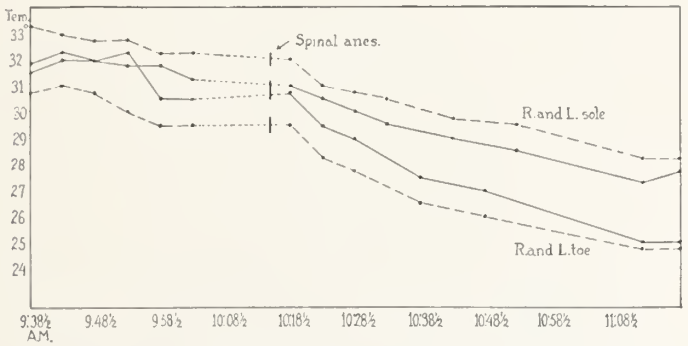

FIG. 4.—(Case 32374.) Unusual response of normal vessels to spinal anesthesia in a few cachectic persons, as shown in a case of carcinoma of the gall-bladder (normal bloodvessels). The temperature does not rise after spinal anesthesia, but starts at a high level.

fell after spinal anesthesia. All 3 of these patients were markedly cachectic and had advanced carcinoma. Apparently their condition had already produced a liberation of the peripheral vessels from all vasoconstriction. We will see this same phenomenon later, also, in certain vascular diseases. These 3 cases of carcinoma are examples of Ipsen's narcosis sign,[9] namely, an unfavorable prognosis in individuals with presumably normal peripheral bloodvessels when the skin temperature in the foot does not rise under general anesthesia. We felt that further information in regard to vasoconstrictor activity could be obtained by studying the effect of other forms of anesthesia. Surgeons have long known that there is a peripheral vasodilatation under general anesthesia. This phenomenon has

been extensively studied by Ipsen,[10] using a simple scheme which shows the major changes.

We wanted to get further evidence, however, in regard to the level and the mechanism of this dilatation. Does general anesthesia produce the same complete relaxation of vasoconstriction as seen following spinal anesthesia? We soon found that nitrous oxide oxygen, ether and tribromethanol, either alone or in sequence, produced the same characteristic sharp increase in the surface temperatures of the feet that were observed under spinal anesthesia.* (Fig. 5.) Also, after about twenty minutes to one-half hour the elevation of the skin temperature reached and held the normal

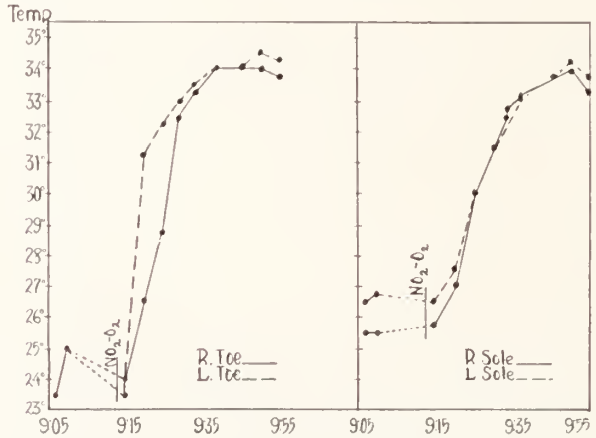

FIG. 5.—Obliteration of normal vasoconstriction by nitrogen monoxide oxygen in a case in which herniotomy was performed on the right side.

vasodilatation level. Fig. 6 shows how the surface temperature of the hand, the sole of the foot and the plantar surface of the great toe all come to within one degree of each other; the vasomotor gradient of the extremity has been obliterated. Furthermore, after this level has been attained, either by spinal anesthesia or by one inhalation agent, the addition of another anesthetic does not alter the surface temperature. (Figs. 7 and 8.) Here we have additional evidence that when once the vasodilatation level is reached the vessels are devoid of vasoconstrictor influence. There are a few

* A preliminary report of the effect of general anesthesia on vasoconstriction was given before the Society for Experimental Biology and Medicine.

sources of error to avoid in testing for this normal vasodilatation level. In the first place, the anesthesia in the affected area must be complete if the nerve fibers are blocked chemically, as in spinal

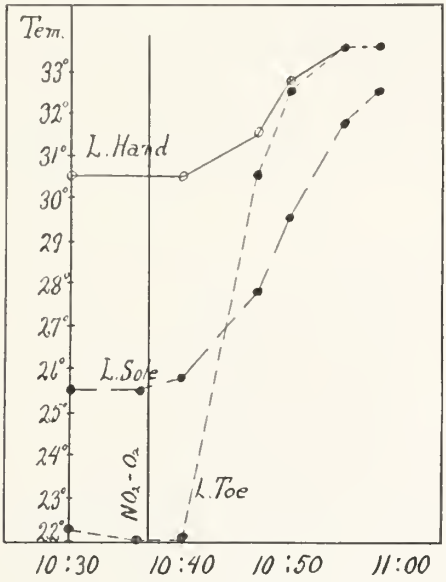

FIG. 6.—(Case 35082.) Normal vessels. All parts of the extremities come to the normal vasodilatation level (obliteration of the vasoconstrictor gradient).

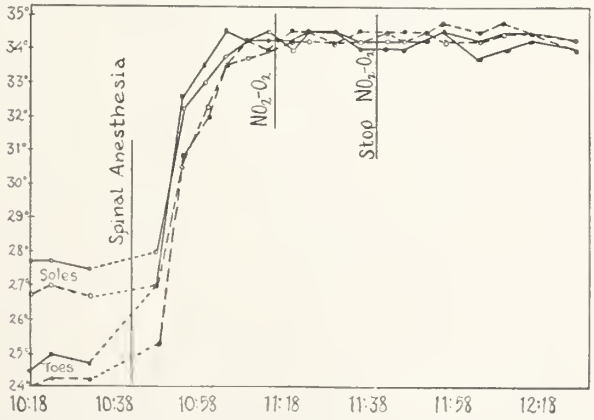

FIG. 7.—When the normal vasodilatation level is obtained by spinal anesthesia the addition of an inhalation anesthetic produces no further effect, as shown in a case in which exploratory laparotomy and ventral herniotomy were performed.

anesthesia. In the second place, under general anesthesia a sufficient depth must be attained to provide muscular relaxation. (Figs. 9 and 10.) In the third place, occasionally some local abnormality of the skin, such as a scar or an extensive callus, will interfere with the normal vascular response at that point. In

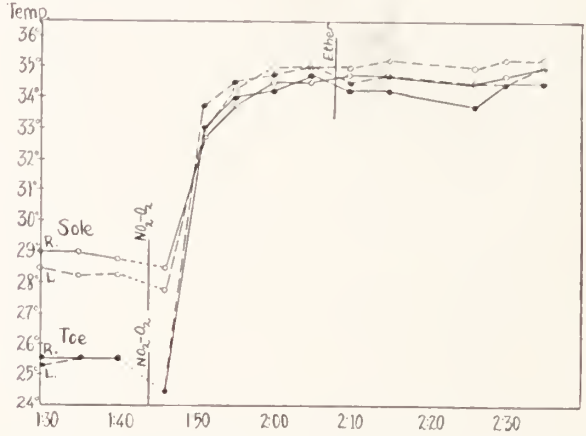

FIG. 8.—When the normal vasodilatation level is obtained by one inhalation anesthetic the addition of another produces no further effect, as demonstrated in a case in which exploration of the wrist was done.

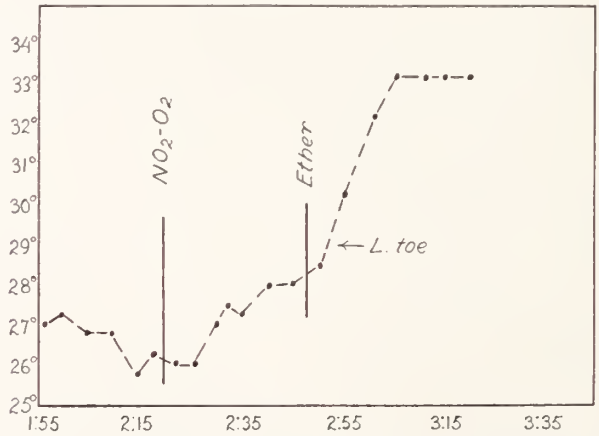

FIG. 9.—(S. B., Case 24432.) Depth of general anesthesia necessary to provide muscular relaxation. Relaxation was not obtained in this case by nitrogen monoxide oxygen alone. Note the unsatisfactory effect on vasoconstriction. On the addition of ether, with relaxation, note the prompt response of the skin temperatures to the normal vasodilatation level. The room temperature was 25.4° C.

fact, where the skin is especially thick the curve of temperature changes is a little slow in rising to its maximum point. Thus, often the temperature of the toe reaches the vasodilatation level more promptly than that of the sole of the foot. When this work was started the surface temperature was determined by a mercury thermometer so constructed that the sensitive surface was flat and applied to the skin. The bulb of the thermometer was covered with cotton or felt. This is similar to the procedure that Ipsen uses. This was sufficient to give the major swing of the temperature, but did not accurately measure the surface temperature itself,

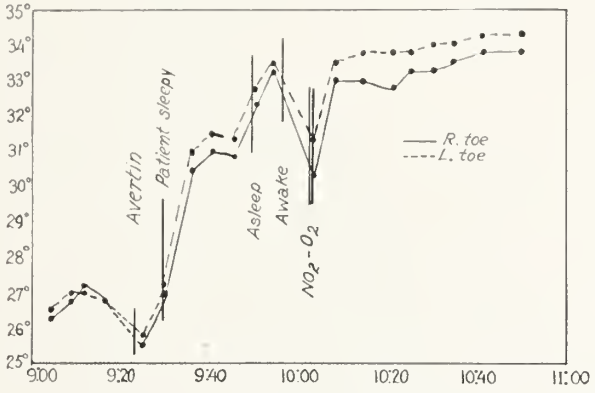

FIG. 10.—(C. B., Case 33971.) Normal vessels; release of vasoconstriction by anesthesia with tribromethanol (in amylene hydrate solution) in a case in which laparotomy was performed for exploration. When the patient was moved to the operating table he awakened, with the recurrence of some vasoconstriction. Nitrogen monoxide oxygen was then administered, with a prompt return to the vasodilatation level. The room temperature was 25.4° C.

due to the retention of heat underneath the felt. The same objection applied also to the usual thermocouple which took from thirty to forty-five seconds to come to equilibrium. Consequently, a simplified and more sensitive apparatus was perfected, requiring about five seconds for each reading.* This interval furnishes a true skin temperature determination without the distortion of heat storage under the apparatus.

By avoiding these errors we then had two methods, either one of which was entirely satisfactory for obliteration of vasoconstrictor

* This apparatus was developed under our direction by the Taylor Instrument Company of Rochester, N. Y., and has been described briefly in the Journal of the American Medical Association.

activity, namely, by spinal anesthesia or by general anesthesia. These left nothing to be desired in studying these reactions in anyone requiring this type of anesthesia for an operation. By this time, however, we had found the test indispensable in the study of any vascular lesion in the extremity. We, therefore, wished to simplify the procedure so that it could be applied with a minimum of discomfort to abulatory patients and could be repeated at intervals without inconvenience. It is well established that the sympathetic motor fibers to the vessels of the extremity are segmentally distributed. Weir Mitchell[13] carried out the experiment of temporarily interrupting impulses through a mixed nerve by freezing his own ulnar nerve and found an immediate increase in temperature in the anesthetized skin field. He also credits Waller with a similar result. Braun,[14] in 1903, emphasizes the vasomotor paralysis with hyperemia and hyperthermia limited to the anesthetic area which follows conduction anesthesia. Wiedhopf[15] made an extensive study of this hyperthermia associated with nerve blocking and advised its use in determining the necessary height for amputation. In fact, he presented evidence suggesting that the vasomotor fibers were more sensitive to the paralyzing action of procaine than the sensory or motor fibers. He also advised freezing the nerve trunks in arteriosclerotic gangrene, not only for the relief of pain but also for the dilatation of the arteries, showing that he had not learned to distinguish the spastic and occlusive elements of vascular disease by this method. Lewis and Landis[16] and White[6] both suggest its application in the qualitative demonstration of arterial spasm. We first investigated the response of normal individuals to such conduction block. Our purpose was to determine how the degree of vasodilatation under such conditions would compare with the normal vasodilatation level previously established. It is obvious that, even if all of the vasoconstrictor impulses in the peripheral field are interrupted by anesthetizing a nerve trunk, vasoconstriction will be present in the larger vessels of the leg. What proportion of the increase in peripheral circulation seen after spinal and general anesthesia will be obtained under peripheral nerve block. This point required elucidation before the method could be used quantitatively to determine spasm and occlusion in pathological cases.

In the majority of instances the problem of peripheral vascular diseases is chiefly concerned with the feet. Consequently, we

wished to produce complete anesthesia in the skin area in the most distal part of the foot. This was easily obtained by infiltration about the posterior tibial nerve a little below the internal malleolus. (Fig. 11.) The nerve at this point can almost always be felt after it has passed under the posterior tibial artery. If novocain is infiltrated in this region conduction block anesthesia of the corresponding skin field involves most of the sole of the foot, the plantar surfaces of the heel and of all of the toes. This procedure was carried out in 16 individuals (medical students) with normal blood-

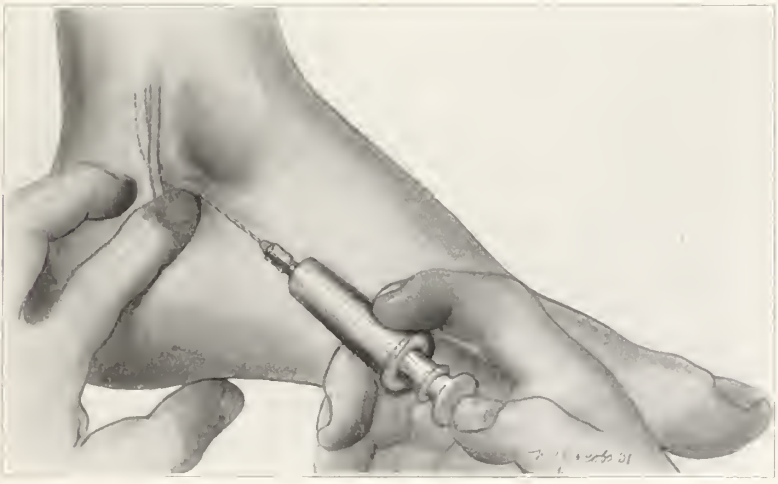

FIG. 11.—Landmarks for posterior tibial nerve block. The nerve is located by palpation and is immobilized with the finger as far distally as it can readily be felt. Injection at this point is distal to the point at which the nerve passes under the posterior tibial artery.

vessels, and it was found that the same form of curve was obtained in the anesthetized area that ensued after spinal or general anesthesia. (Fig. 12.) Under standard conditions the height of the curve in these normal individuals come within a range of 1.5° C., but the mean of maximum temperatures obtained under conduction block was about 1° C. below that obtained under spinal or general anesthesia. Consequently, it seemed clear that in the involved area all but a small fraction of the increased circulation which follows spinal and general anesthesia was obtained by blocking the vasoconstrictor fibers in the nerve trunk only a few inches from the

peripheral field. In order to verify this the maximum surface temperature readings on the foot of the same individual were compared, following conduction block of the posterior tibial nerve, with those obtained shortly thereafter under general anesthesia. (Fig. 13.) The skin temperature of the great toe reached its height at 32.9° C., under the nerve block, while its maximum under nitrous oxide-oxygen-ether anesthesia was 33.6° C. It is apparent

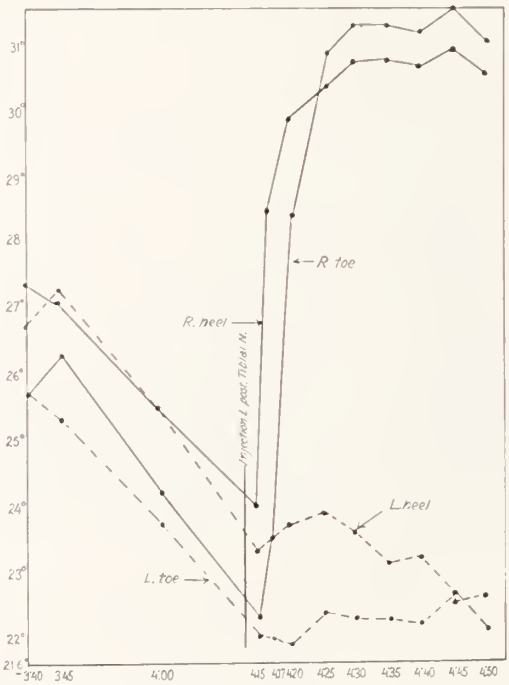

FIG. 12.—Normal vessels, showing vasodilatation obtained following conduction block of the right posterior tibial nerve in R. W. The temperature of the room was 20° C.

then, that conduction block is a satisfactory method of estimating vasoconstriction, though in borderline pathological cases it is probably desirable to check the data so obtained, with the result of either spinal or general anesthesia.

We now see the effect on the surface temperatures of the extremities of temporarily abolishing vasoconstriction by three different methods and have established the normal vasodilatation level by

these procedures, as follows: At a room temperature of 20° C. (68° F.) the surface temperature of the great toe reaches at least 31.5° C. with general or spinal anesthesia and 30.5° C. with nerve block of the posterior tibial nerve. (If the determination is carried out in a warmer room a correction factor of 0.3° C. should be added to these levels for each degree Centigrade by which the room temperature exceeds 20° C.) By comparing the effect obtained in pathological cases with these figures for the normal response we are

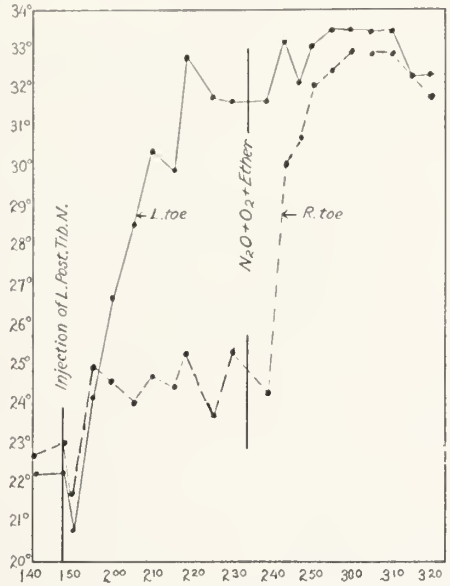

FIG. 13.—Normal vessels; comparison of vasodilatation obtained by blocking the left posterior tibial nerve with that following inhalation anesthesia in J. J. The temperature of the room was 22° C.

now able to divide all peripheral vascular disturbances in the extremities into three main categories. The circulatory symptoms in the respective groups are due to (1) occlusion alone, (2) spasm alone and (3) mixed spasm and occlusion. Most endarteritis obliterans, whether of diabetic or arteriosclerotic origin, fall into the first group of occlusion alone. (Figs. 14 to 17.) Probably most cases of Raynaud's disease and many of the cases that have been classified as thromboangiitis obliterans respond in their earlier stages to anesthetization of the vasoconstrictor fibers with a dilata-

tion to the normal level. (Figs. 18 to 20.) Many examples of thromboangiitis obliterans and also some of syphilitic arteritis show by this test both spasm and occlusion. (Figs. 21 and 22.)

In the first group (occlusion) with the establishment of a satisfactory anesthesia by any of the three methods outlined there is practically no increase in the surface temperature of the involved part. Frequently the most distal part of the extremity will show this type of response while a small amount of vasoconstriction can

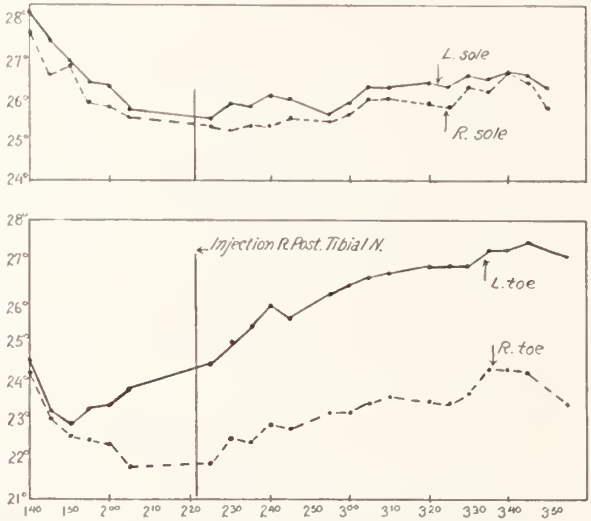

FIG. 14.—Endarteritis obliterans in C. W. No palpable pulses in either foot. Right toes show marked dependent rubor. Gangrene threatened on the right, not on the left. Injection of the right posterior tibial nerve causes no significant increase in temperatures. Gradual increase in surface temperature of left toe occurs on the unanesthetized side. Interpretation: circulatory deficiency in right foot due to occlusion without spasm. Room temperature was 20° C.

still be demonstrated in more proximal areas. The second group (spasm) includes many examples where the history strongly suggests a spastic lesion by the extreme variation of symptoms during different periods. This is, of course, particularly well exemplified by the white and blue phases of Raynaud's disease* with intervening periods of normal color and apparently normal circulation. But many times it is found that spasm is the outstanding cause of

* The arterial spasm of Raynaud's disease is somewhat complicated and is being fully discussed in a separate study (to be published in *Annals of Surgery*.)

symptoms in cases of common vascular diseases that appear clinically exactly like the outspoken members of the occlusion group. A large number of the presenile vascular diseases show both spasm and occlusion. Obviously there are many degrees in the exact proportion of each of these two elements among the various cases. Some show a mere trace of spasm still present, while others attain

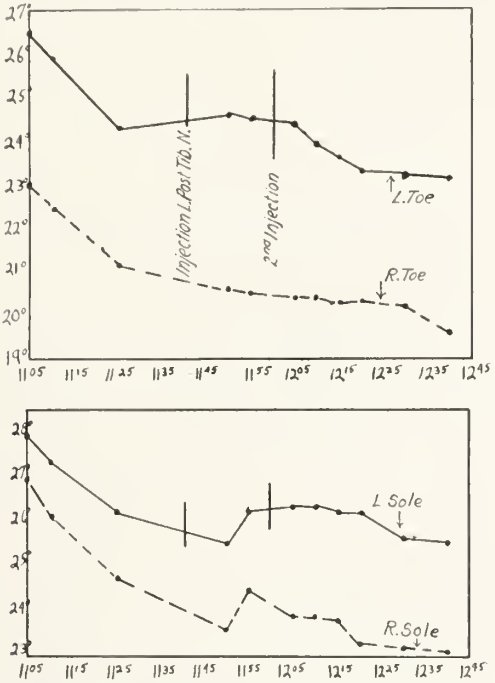

FIG. 15.—Endarteritis obliterans in a man, aged forty-one years. Circulation in the left foot dangerously impaired, with a small indolent ulcer on the dorsal surface of the left great toe. Conduction block of left posterior tibial nerve was not followed by increase in temperature, indicating occlusion without spasm. Temperature of the room was 21.1° C.

almost the normal levels after anesthesia. To designate the proportions of spasm and occlusion in any area, the highest surface temperature attained (in degrees Centigrade) is subtracted from the lowest normal reading established for the type of anesthesia used. This figure is called the *occlusion index* and is of importance in determining the type of treatment to be adopted in the mixed group. We may emphasize here the fact that the above clinical

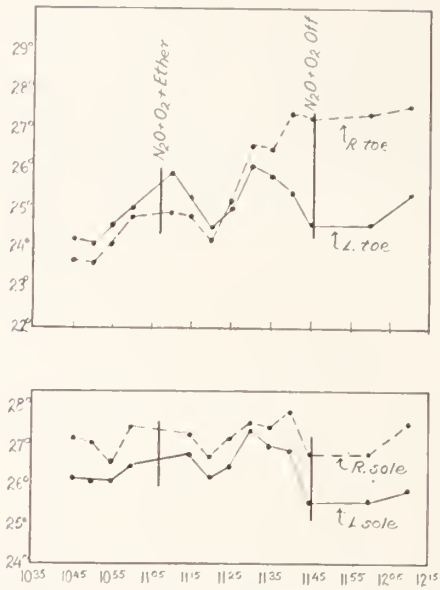

FIG. 16.—Diabetic endarteritis obliterans in Mr. E. S. No increase in surface temperature of the left toes; those on the right show very slight increase with an occlusion index of 5° C. The temperature of the room was 21.1° C.

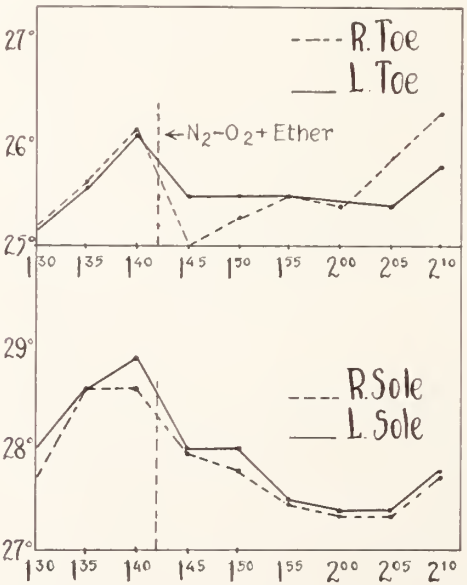

FIG. 17.—(Case 6756.) Diabetic endarteritis obliterans. No increase in surface temperature after inhalation anesthesia. Occlusion without spasm. Temperature of the room was 21.1° C.

groups are established upon the basis of their physiological response rather than upon a pathologico-anatomical basis. Probably a certain amount of organic occlusion by thrombosis or luminal narrowing is present in nearly all of the cases of vascular disease with the exception of the earlier stages of Raynaud's disease. But there is a very important difference in the effect of such changes upon the

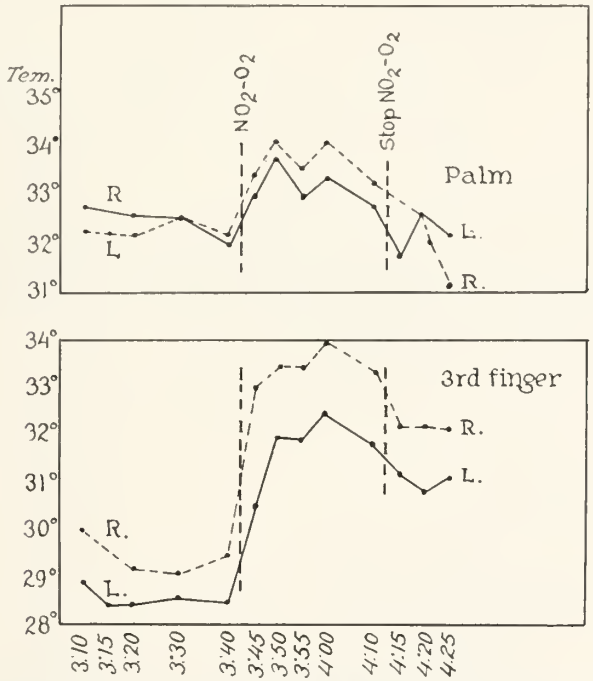

FIG. 18.—Angiospasm of the hands (Raynaud's type). Following inhalation anesthesia the right third finger reaches the vasodilatation level. The left shows a marked increase, but remains nearly 2 degrees below the right, showing a slight occlusion index in the left third finger. This corresponds with the clinical picture, as there had been a superficial area of necrosis on the left third finger, leaving a scar.

circulation of the foot, primarily due to the behavior of the collateral and terminal vessels. The location of these important arteries is visualized in these roentgenograms. Fig. 23 shows an *intra vitam* injection of the arterial tree with sodium iodid. An obstruction in the posterior tibial artery is seen, the small anastomotic branches running parallel to the main vessel can be followed down to where the channel is reconstituted in the main artery about

opposite the internal malleolus. Fig. 24 is an injected specimen of a leg, amputated on account of infection. It shows very nicely in the foot, the smaller terminal arteries. In advanced noninflammatory occlusive disease, such as that typically seen in arteriosclerosis or diabetic endarteritis, vasoconstriction, even that amount usually present in normal individuals is lacking. Apparently some compensatory mechanism has released the anastomotic and ter-

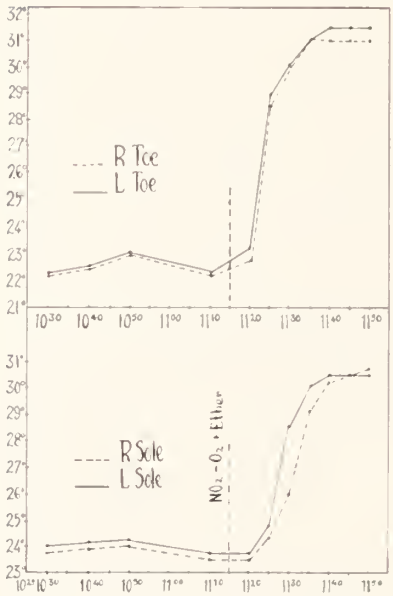

FIG. 19

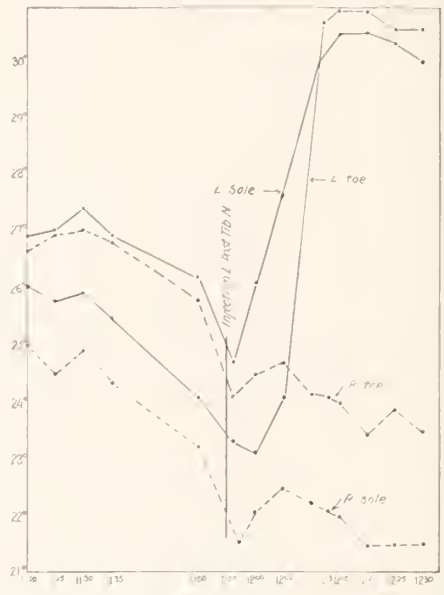

FIG. 20

FIG. 19.—(Case 42685.) "Diabetic endarteritis obliterans," clinical diagnosis. Circulatory impairment causes pain and disability, more severe on the right. Test with inhalation anesthetic shows circulatory symptoms chiefly due to spasm. Occlusion index about 1° C. The room temperature was 22.7° C.

FIG. 20.—Same effect shown by conduction block of left posterior tibial nerve in a case of arteriospasm in F. C. S. Compare the heights of the temperatures obtained under nerve block and inhalation anesthesias. Temperature of the room was 20° C.

minal arteries and arterioles from all vasoconstrictor effects. (Figs. 25 and 26.) It is exactly here that vascular disease associated with spasm differs from the occlusive form. In advanced stages of arteriospasm threatening the viability of the part vasoconstriction still persists and is not released by the compensatory mechanism seen in the occlusive group. What causes this difference? We do not yet have final proof, but it is our hypothesis that the inflamma-

tory reaction extending through the wall of the vessels, which is so characteristic pathologically of thromboangiitis obliterans, affects the afferent nerve fibers in the adventitial coat or even in the accompanying nerves themselves. This sets up a reflex vasocon-

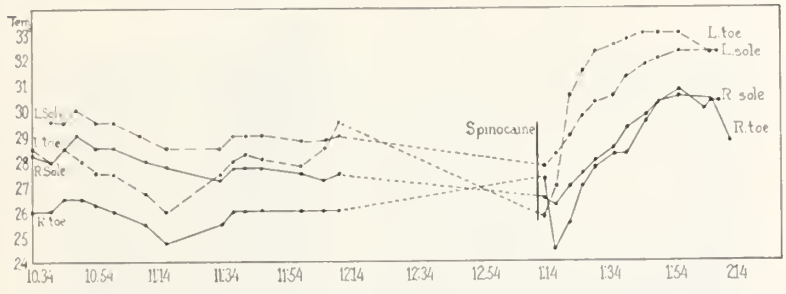

FIG. 21.—(Case 31391.) Syphilitic endarteritis obliterans. No pulsations in the right foot and feeble pulsations in the left. Circulation seriously impaired on the right. Following spinal anesthesia the left foot comes up to the normal vasodilatation level. Right foot shows a definite but incomplete rise with an occlusion index of 2.5° C.

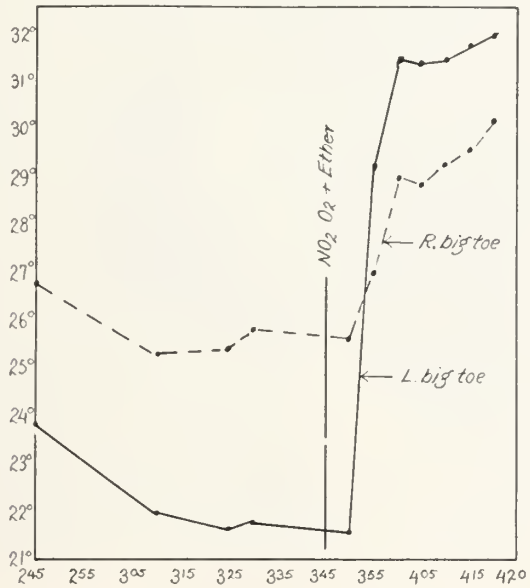

FIG. 22.—Thromboangiitis obliterans with migratory phlebitis in P. B. Symptoms at present limited to the right. Inhalation anesthesia shows response on the left to the normal vasodilatation level; on the right a definite but incomplete rise. Occlusion index, 2° C. Temperature of the room was 21° C.

strictor spasm, the particular importance of which is that it involves not only the diseased main arteries but also the collateral and terminal small vessels in the periphery. Consequently, when the circulatory deficit reaches a certain point, in occlusion it brings

FIG. 23.—Arteriosclerosis with occlusion of posterior tibial artery; *intra vitam* injection. This figure is shown to illustrate the collateral circulation through the small vessels. The posterior tibial artery blocked in about the middle of the calf is reconstituted opposite the malleolus (retouched photograph).

into play the compensatory release of normal vasoconstriction, but in the spastic group this reflex vasoconstriction continues. We have found that in vascular disease the efficiency of the circulation to the distal part of the extremity is much more dependent upon

these smaller arteries than it is upon the main arterial trunk. An illustration of this is found in the following case—one of arteriosclerotic endarteritis with diminution or absence of pulsation in all four major vessels of both feet. However, the left dorsalis pedis artery could barely be felt. The right was not palpable. This

FIG. 24.—Injection of amputated extremity to illustrate the terminal finer arteries. Note the occlusion of the posterior tibial artery in the lower third of the leg shown between the tibia and fibula. The viability of the foot is more dependent on the anastomotic and terminal arteries and arterioles than on the main arteries in the lower leg.

greater impairment of the large vessels in the right lower leg was borne out by the following oscillometric readings. On the left side the swing was $1\frac{1}{2}$ major divisions, while on the right side it was only $\frac{1}{2}$.

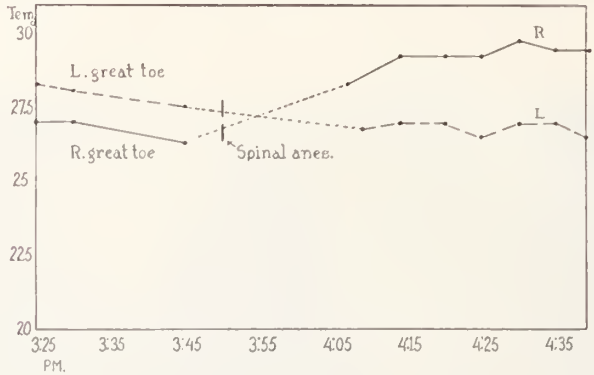

FIG. 25.—(Case 31766.) Endarteritis obliterans on the left. No pulsation on the left, feeble on the right. Circulation of left foot dangerously impaired. Left great toe shows no rise after spinal anesthesia; right great toe shows slight increase. Note that the side with the more seriously involved peripheral circulation starts at a higher level and shows no vasoconstriction, while the less involved side shows a slight amount of vasoconstriction still present.

Yet in spite of the more complete occlusion of the larger arteries, the temperatures of the right toes were 1 degree higher than those on the left, showing a more rapid peripheral circulation on the side with the more defective major arteries. This phenomenon was

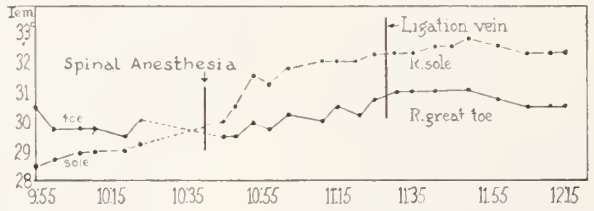

FIG. 26.—(Case 26956.) Diabetic endarteritis obliterans (arteriosclerotic vessels). Left leg amputated previously. Circulation in right toes seriously impaired. Following spinal anesthesia no increase in surface temperature of the toes, but definite increase in the sole. Note particularly the reversal of the normal vasoconstrictor gradient, with the temperature of the toe starting at a higher initial level than that of the sole, owing to the absence of vasoconstriction in the toe.

probably due to the fact that the left side was still affected by vasoconstriction, while on the right side the compensatory mechanism had released it.

In reviewing the relationship of sympathetic stimuli to peripheral bloodvessels, we have then a varying degree of vasoconstrictor activity in the normal individual. In occlusive disease without any inflammatory reaction in the outer wall of the bloodvessels this normal vasoconstriction is released when the circulation becomes seriously impaired. In spastic disease vasoconstriction continues even when the reduced circulation to the part endangers its viability. When some of the anastomotic and terminal vessels also become occluded then we have the mixed type. There are many more examples of spasm among the common diseases of peripheral bloodvessels than is usually realized.

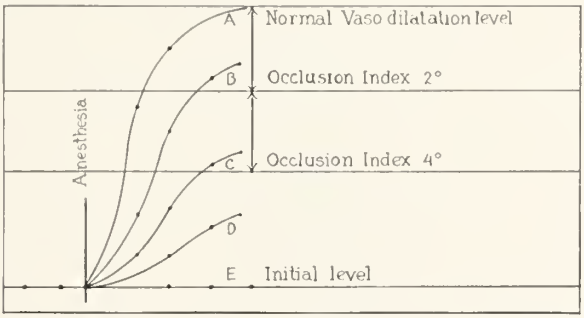

FIG. 27.—Response to anesthesia as a guide to treatment. If the surface temperature rises to the normal vasodilatation level, curve *A*, or to within 2° C. (3.6° F.) of this point (occlusion index less than 2° C.), curve *B*, the treatment should be directed toward overcoming the spasm. If the rise in temperature is definite with an occlusion index between 2° and 4° C. (3.6° to 7.2° F.), curve *C*, the release of spasm may be of assistance. But if the rise in surface temperature is only to a point at which the occlusion index is more than 4° C., curve *D*, or if there is no rise, curve *E*, treatment should be for occlusion, sympathectomy is unjustifiable.

TREATMENT. (Fig. 27.) The differentiation of occlusive and spastic vascular disease and the estimation of the relative proportion of each when both are present is of prime importance because the principles upon which the treatment of these groups must rest differ radically. In the group whose symptoms are entirely dependent upon occlusion nothing whatever is gained by elimination of vasoconstrictor activity. Sympathectomy for such individuals is unjustifiable. Besides symptomatic relief of discomfort and the avoidance of infection, treatment should be directed to only one end, namely, a more efficient peripheral distribution of the blood. Venous ligation seems to offer some assistance in this direction. It is probably in this manner that pain in such cases may be somewhat

alleviated by depressing the level of the feet, thereby using gravity to assist in the forcing of blood through the most distal parts. It is not our purpose here to discuss extensively the treatment of occlusion. We want particularly to emphasize that there is no evidence of participation of the sympathetic activity in this group. On the other hand, in those conditions which show spasm the treatment is entirely different and should consist of measures to overcome the spasm present. There are several ways in which this problem may be attacked.

1. The most direct and satisfactory procedure would be to overcome the inflammatory process. In syphilitic vascular disease this can sometimes be achieved, and the possibility of this specific agency being the cause of the condition should always be considered, particularly in the mixed group showing both spasm and occlusion. It is our impression, from the limited number of examples of syphilitic arteritis in which we have tested for spasm, that the latter was mixed with some occlusion and was not usually an especially striking phenomenon. In the arterial spasm of thromboangiitis obliterans and Raynaud's disease we do not yet know the primary cause of the reaction. Any information along this line will be very gratefully received by all of us who are required to treat vascular diseases in the extremity.

2. *Vasodilator Drugs.* It is natural that we should next turn for assistance to the pharmacological agents which cause vasodilatation or oppose sympathetic stimulation. We have tried out in this connection the nitrites, acetyl choline and ergotamine, but we have not been able to obtain an immediate effect on the peripheral vessels of the extremity in arterial spastic disease that at all approaches the interruption of the sympathetic fibers.

3. *Local Heat.* In certain forms of arterial spasm heat applied locally relaxes the spasm. This is particularly true in certain traumatic lesions. It is impossible to use surface temperature readings directly as an index of the effect of local heat on the vascular circulation. We have studied in isolated instances the normal recovery from heat and cold, but have not yet established satisfactory criteria for the normal limits. The effect of local heat in arterial spastic disease is not as lasting as the application of heat to raise the body temperature.

4. *General Heat.* We have produced hyperthermia in these patients by two methods, (*a*) diathermy and (*b*) hot baths. Dr. S. L.

Warren supervised the diathermy treatments which consisted in the passage of 5 amperes of high-frequency current (wave length, 300 meters) through the body between large electrodes. The continuous hot baths were given by the method recently advised by Mehrtens and Ponppirt.[17] In our cases both procedures caused a relaxation from arterial spasm which outlasted the hyperthermia

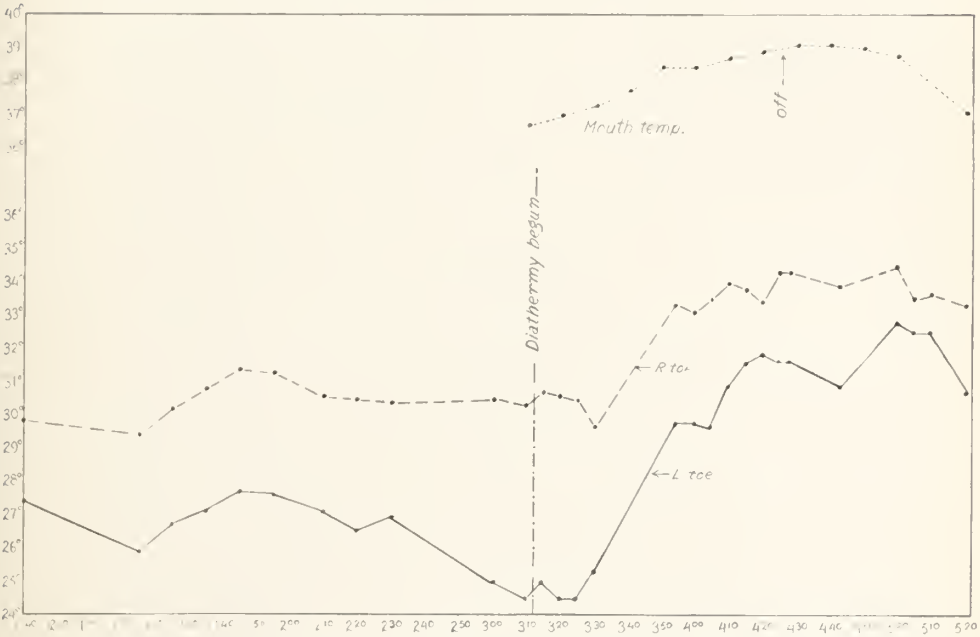

FIG. 28.—Arteriospasm, the effect of diathermy in Mr. C. B. Sympathectomy had been performed on the right side. Diathermy caused a rise in the mouth temperature and the surface temperature of the denervated side, but a much greater rise proportionately on the side not operated upon. A difference of over 5° C. (9° F.) in the skin temperature of the two toes recorded at the beginning of treatment is reduced to 1° C. (1.8° F.). Note also that the surface temperature of the left toe is still elevated considerably above its initial point after the mouth temperature had returned to normal. Temperature of the room was 22° C.

produced. (Figs. 28, 29 and 30.) Pain is temporarily alleviated. We have not yet followed these patients long enough to determine how effective these measures will prove in permanently keeping the spasm under control. We believe, however, that they are a much more desirable form of hyperthermia than that following foreign protein.

5. *Foreign Protein.* The injection of various types of foreign protein has long been used in the treatment of several conditions, among them peripheral vascular diseases. Allen and Smithwick[18] and Brown and Henderson[19] have found considerable improvement following repeated injections of typhoid vaccine. The former workers have called attention to the danger of thrombosis with this method.[5] Even though the effect may prove somewhat more per-

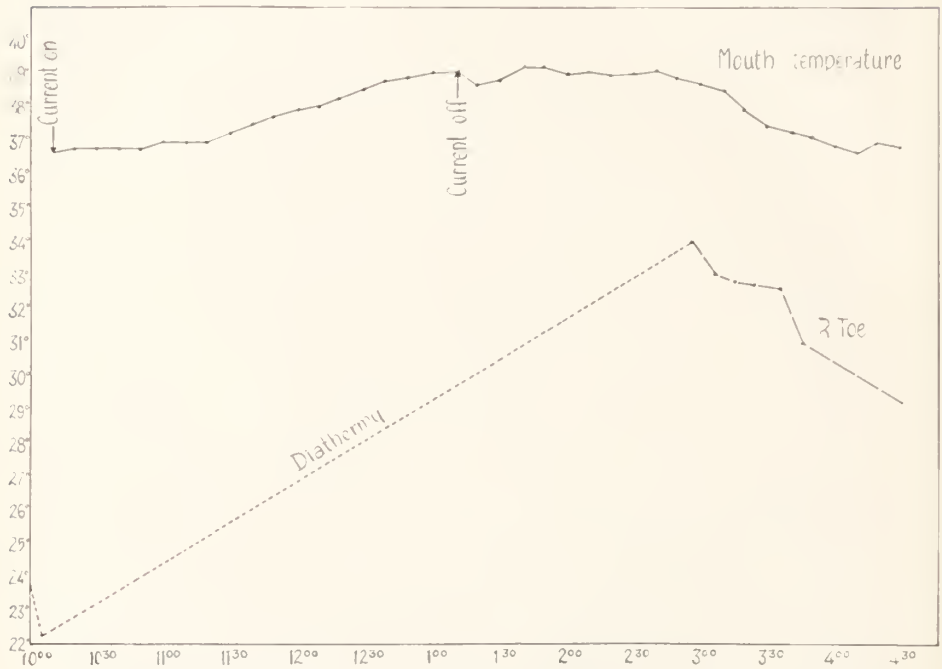

FIG. 29.—Arteriospasm, the effect of diathermy in Mr. F. S. Note that the surface temperature in the toes is still elevated three hours after the diathermy was discontinued and after the mouth temperature had returned to normal. Temperature of the room was 25° C.

sistent than that following hyperthermia produced by the methods described above, the severity of reactions whether or not associated with complications makes it an undesirable form of treatment.

6. *Surgical Interruption of Sympathetic Fibers.* Whenever vasoconstriction is demonstrated it can be overcome by the interruption of the sympathetic fibers to the involved area. This may be done at different levels. The first attempt to accomplish this was peri-

arterial sympathectomy. Leriche developed the procedure of denervating a length of artery by removal of its adventitia. He thought that thereby he interrupted efferent fibers to the peripheral field. Following this procedure it was reported that a vasodilatation occurred in the corresponding extremity. Later it was added that vasodilatation also occurred in other extremities which was

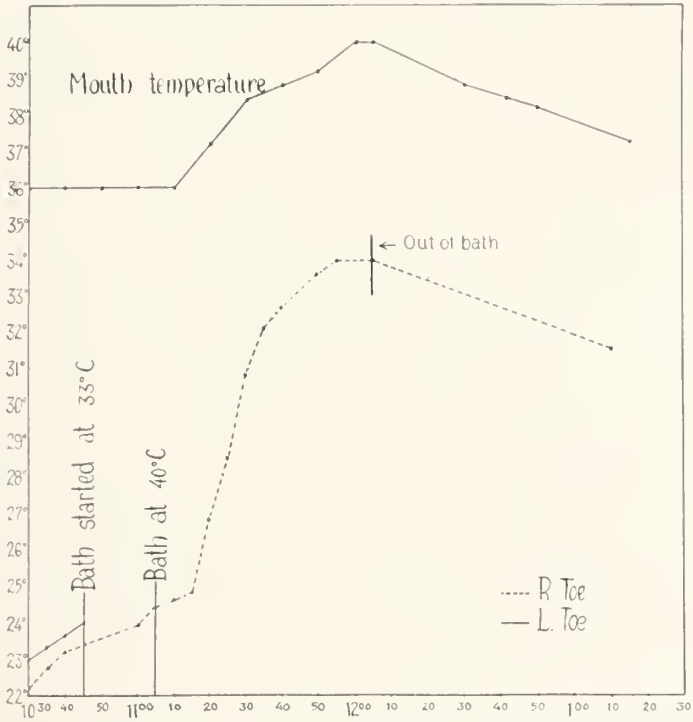

FIG. 30.—Arteriospasm, the effect of a continuous hot bath in Mr. F. S. Note the more rapid fall in mouth temperature than in surface temperature after the completion of the bath. Temperature of the room was 22.7° C.

thought due to some type of reflex disturbance. More recently anatomical studies have verified the previous anatomical and physiological conception that the vasoconstrictor fibers for distant fields are confined to the main nerve trunks. A study of the surface temperatures in the extremities after other simple operations, such as herniotomy, shows that some of these vascular changes previously thought due to interruption of sympathetic fibers are merely non-

specific postoperative effects. (Fig. 31.) Possibly some of the results reported are due to interrupting afferent fibers. Under any circumstance it is quite clear that periarterial sympathectomy is not a satisfactory method of overcoming vasoconstrictor spasm in the extremities.

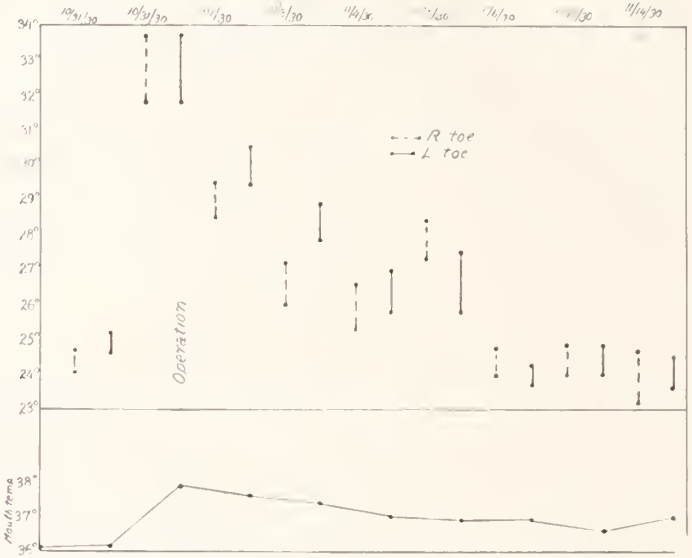

FIG. 31.—Nonspecific postoperative effect on peripheral circulation of normal vessels in C. M., upon whom herniotomy had been performed on the right side. Each line represents the maximum and minimum temperatures recorded under standard conditions (room temperature, 20° C.) for a period of one-half hour each day. Note the marked elevation above the original level for several days following herniotomy under local anesthesia.

Ganglionectomy. Approximately the same degree of vasodilatation which can be temporarily induced by anesthesia can be made permanent by sympathetic ganglionectomy in the proper area. The duration of the effect has been questioned. We have cases now that have maintained a constant elevation to a high level on the operated side for more than two years. (Figs. 32 and 33.) Brown and Adson,[20] Filatov[21] and others report similar effects, some over even a longer period of time. The influence of sympathectomy on the progress of the thrombotic process in the mixed group, such as in certain cases of thromboangiitis obliterans, is not known as yet. It is our belief that not only will the circulation be relieved of the

spastic element but also the absence of spasm may exert a beneficial effect to retard extension of the thrombosis. Our opinion in this matter is based upon the known development of thrombosis as a

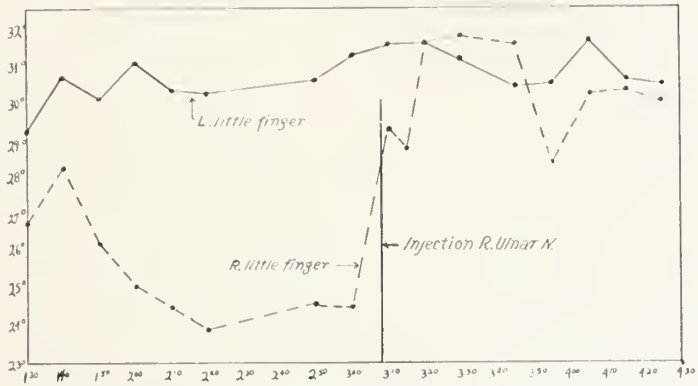

FIG. 32.—Left cervical sympathectomy three months previous to test in Miss L. D. Surface temperature of the left fingers was maintained at a high level, which is the same level reached by the right ittle finger after the right ulnar nerve was blocked with procaine hydrochloride. Temperature of the room was 20.5° C.

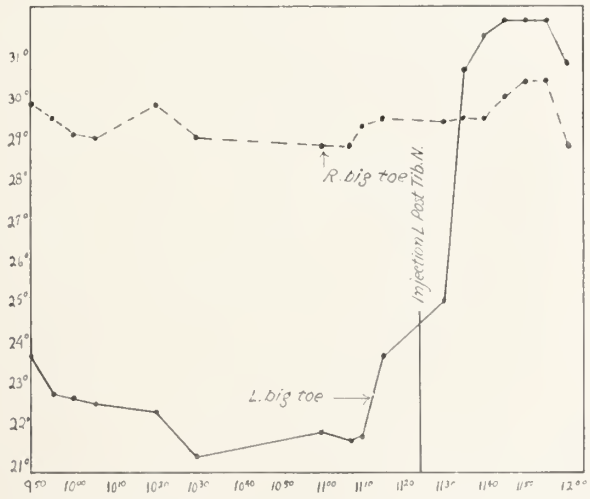

FIG. 33.—Thromboangiitis obliterans twenty months after right lumbar sympathectomy in P. B. Surface temperature on the right side has been maintained at a high level continuously since operation (for twenty months when this paper was written), together with great improvement in symptoms on this side. The level maintained is a little below that reached by the left great toe after the left posterior tibial nerve was blocked with procaine hydrochloride. Temperature of the room was 20° C.

complication of the arterial spasm in the later stages of Raynaud's disease. It is also influenced by the possibility that reflex spasm of the vasovasorum may be a factor in an extension of the disease. These considerations in regard to the further progress of the thrombotic element are at present theoretical; the effect of ganglionectomy in improving the peripheral circulation by removing arterial spasm is proven.

In some cases, particularly where pain is an important element in the disease, as Smithwick and White[22] have pointed out, peripheral nerve interruption may be of benefit to overcome the pain and to release the arterial spasm in the peripheral area supplied. This is particularly applicable to older individuals who should not be submitted to ganglionectomy and where hyperthermia by physical means is not advisable or proves insufficient. We have recently used alcohol injection of the peripheral nerve with great satisfaction in overcoming the painful reaction to cold in a case of causalgia.

In the mixed group we do not yet have a sufficiently extensive experience to decide definitely the proportion of occlusion beyond which the results do not justify sympathectomy. As a tentative plan, we are not doing this operation if the occlusion index is over 2° C. in the sole. We have seen improvement in the circulation of the foot after sympathetic denervation with the progression of the thrombotic process in one or two toes to the point that local amputation of the latter was necessary. This we did not consider a reflection on the desirability of the sympathectomy.

In brief, as long as spasm is the dominant factor in the viability of the extremity measures are mainly directed toward the overcoming of this vasoconstriction. We know that surgical interruption of the sympathetic fibers will effect this result promptly. Certain simpler methods, however, also do so temporarily. The exact value of the latter must be worked out by extensive studies. At present the choice between conservative measures and an operation on the sympathetic system must be decided in each case individually. If spasm cannot be controlled by other measures sympathetic ganglionectomy may be less radical than nonoperative treatment.

This study of the sympathetic influence on the vascular system has brought to light a fundamental difference in the control of the peripheral and the splanchnic arteries. The usual surgical depth of general anesthesia entirely obliterates vasoconstriction in the

extremities, while it scarcely affects that of the splanchnic area. This seems to be dependent on an anatomical or, more probably, a phylogenetic difference in the vasomotor control for these regions.

In the future data based upon this distinction of spasm and occlusion and a more minute study of these elements will greatly assist in settling many fundamental questions concerning peripheral vascular diseases. When and how in the occlusive group does obliteration of normal vasoconstriction occur? How does the arteriospasm of the spastic group differ from normal vasoconstriction—in severity, in persistence or in some other way?

SUMMARY. Pioneers have recently opened up important possibilities in the application of our anatomical and physiological knowledge of the sympathetic nervous system to several clinical syndromes. For the achievement of sound progress in this interesting field we now need the establishment of criteria, by which to measure the sympathetic activity in the various systems. The effect of temporarily interrupting the extrinsic nerve supply to the large bowel by spinal anesthesia is offered as an index of the amount of sympathetic inhibition present in megalocolon. For vascular diseases of the extremities a simple, systematic test is outlined which distinguishes the effects of spasm and of occlusion. The vasoconstrictor stimuli to the area studied are interrupted by anesthesia, the effect of which on the surface temperature is compared with the similar response of normal individuals. We have established the normal levels for spinal, general and conduction block anesthesia. In an individual case if anesthesia is accompanied by no elevation of temperature in the cool distal part of the extremity then occlusion without spasm is present; if the skin temperature reaches the normal vasodilatation level then sympathetic vasoconstriction is responsible for the circulatory symptoms; if the elevation is definite but not to this level then both occlusion and spasm participate. The difference between the highest temperature obtained and the normal vasodilatation level has been termed the occlusion index, as it measures the effect of organic obstruction to the peripheral circulation. Nerve block anesthesia is particularly useful in this vasomotor test on account of its simplicity and freedom from discomfort. In doubtful or borderline cases its results should be checked with those of spinal or general anesthesia.

By these methods valuable information in regard to the nature and the treatment of various types of vascular disease in the extremi-

ties is obtained. In the purely occlusive group some compensatory mechanism releases the collateral and terminal vessels from vasoconstriction when the local circulation is seriously reduced. This does not obtain in the spastic group.

The treatment of these two groups should be along radically different lines. The principle of that for purely occlusive disease is the more peripheral distribution of the reduced amount of blood available. While in spastic disease the treatment should be directed toward an augmentation of the local circulation, by overcoming sympathetic vasoconstrictor spasm. Particularly valuable methods for this are hyperthermia producing temporary vasodilatation, sympathetic ganglionectomy for permanent effects and peripheral nerve interruption where ganglionectomy is desirable but contraindicated by the patient's condition.

BIBLIOGRAPHY

1. Scott, W. J. M., and Morton, J. J.: Sympathetic Inhibition of the Large Intestine in Hirschsprung's Disease, Jour. Clin. Invest., 1930, ix, 247.

2. Brown-Sequard, C. E.: Course of Lectures on the Physiology and Pathology of the Central Nervous System. Delivered at the Royal College of Surgeons of England, May, 1858, Philadelphia, 1873, p. 148.

3. Babinski, J., and Heitz, J.: Obliterations arterielles et troubles vasomoteurs d'origine reflex ou centrale, Bull. et mém. Soc. méd. d. hôp. de Paris, 1916, xl, 570.

4. Brown, G. E.: The Treatment of Peripheral Vascular Disturbances of the Extremities, Jour. Am. Med. Assn., 1926, lxxxvii, 379.

5. Allen, A. W., and Smithwick, R. H.: Thrombosis of Peripheral Arteries Following Intravenous Injection of Typhoid Vaccine: Report of Two Cases, New England Jour. Med., 1929, cc, 217.

6. White, J. C.: Diagnostic Blocking of Sympathetic Nerves to Extremities with Procaine, Jour. Am. Med. Assn., 1930, xciv, 1381.

7. Brill, S., and Lawrence, L. B.: Changes in Temperature of the Lower Extremities Following the Induction of Spinal Anesthesia, Proc. Soc. Exp. Biol. and Med., 1930, xxvii, 728.

8. Morton, J. J., and Scott, W. J. M.: The Measurement of Sympathetic Vasoconstrictor Activity in the Lower Extremities, Jour. Clin. Invest., 1930, ix, 235.

9. Foged, J.: Ipsen's Narkosephanomen, Deutsch. Ztschr. f. Chir., 1930, ccxxix, 365.

10. Ipsen, J.: Les Arteres et l'anesthesie, Acta chir. scand., 1929, vi, 487.

11. Scott, W. J. M., and Morton, J. J.: Obliteration of Vasoconstrictor Gradient in the Extremities Under Nitrous Oxide Oxygen, Ether and Tribromethyl Alcohol Anesthesias, Proc. Soc. Exp. Biol. and Med., 1930, xxvii, 945.

12. Scott, W. J. M.: An Improved Electrothermal Instrument for Measuring Surface Temperatures, Jour. Am. Med. Assn., 1930, xciv, 1987.

13. Mitchell, S. Weir: Cases of Lesions of Peripheral Nerve Trunks with Commentaries, Am. Jour. Med. Sci., 1883, lxxxvi, 17.

14. Braun, H.: Experimentelle Untersuchungen und Erfahrungen über Leitungsanaesthesie, Arch. f. klin. Chir., 1903, lxxi, 179.

15. Wiedhopf, O.: Experimentelle Untersuchungen über die Wirkung der Periarteriellen sympathectome und der Nervenvereisungen auf die Gefasse der Extremitaten, Beitr. z. klin. Chir., 1924, cxxx, 399.

16. Lewis, T., and Landis, E. M.: Some Physiological Effects of Sympathetic Ganglionectomy in the Human Being and Its Effect in a Case of Raynaud's Malady, Heart, 1930, xv, 151.

17. Mehrtens, H. G., and Pouppirt, P. S.: Treatment of Intermittent Claudication With Hyperpyrexia Produced by Baths, Jour. Am. Med. Assn., 1930, xcv, 1910.

18. Allen, A. W., and Smithwick, R. H.: Use of Foreign Protein in Treatment of Peripheral Vascular Diseases: Results of Intravenous Injections of Typhoid Vaccine, Jour. Am. Med. Assn., 1928, xci, 1161.

19. Brown, G. E., and Henderson, M. S.: The Diagnosis and Treatment of Arterial Vascular Disease of the Extremities, Jour. Bone and Joint Surg., 1927, ix, 613.

20. Brown, G. E., and Adson, A. W.: Physiologic Effects of Thoracic and of Lumbar Sympathetic Ganglionectomy or Section of Trunk, Arch. Neurol. and Psychiat., 1929, xxii, 322.

21. Filatov, A.: Klinische und experimentelle Untersuchungen über die Schwankugen der Haut Temperature nach Chirurgischen Eingriffen am lumbalen und cervicalen Abschnitte des sympatheschen nervensystems, Beitr. z. klin. Chir., 1930, cxciv, 95.

22. Smithwick, R. H., and White, J. C.: Elimination of Pain in Obliterative Vascular Disease of the Lower Extremity (A Technique for Alcohol Injection of the Sensory Nerves of the Lower Leg), Surg., Gynec. and Obst., 1930, li, 394.

THE COMMUNITY HEALTH PROGRAM*

By C.-E. A. WINSLOW, Dr. P.H.,
PROFESSOR OF PUBLIC HEALTH, YALE SCHOOL OF MEDICINE, CHAIRMAN; COMMITTEE ON ADMINISTRATIVE PRACTICE, AMERICAN PUBLIC HEALTH ASSOCIATION

Premonitions. We may take as our text for this evening the saying of Hippocrates—"Where there is love for humanity, there also is love for the art of medicine." Every organized health program is an illustration of this saying—an expression of the desire of mankind that the fruits of the tree of healing may be made available to all. Such a program is often initiated from outside the medical profession, from those who desire service rather than from those who provide service. Sometimes its trends may run counter to what the physician himself believes to be sound policy. In its essence, however, every organized effort toward community health is a profound tribute to the professions of medicine, of nursing, of bacteriology, of sanitary engineering, of social service—whose benefits it demands in richer degree and fuller measure.

From very early days there have been efforts to develop an organized defense against preventable disease. The half sanitary and half religious ritual of the Persian Zendavesta with regard to pollution of water and earth and avoidance of contact with dead bodies represented a primitive foreshadowing of a community health program, as, indeed, did the taboos and charms and amulets and fetiches and ritual dances of much more primitive peoples. The Book of Levitieus marks a tremendous advance toward a rational prevention of contagion by isolation and disinfection. As Neuburger says, the chief glory of Biblical medicine lies in the institution of social hygiene as a science.

In regard to the control of communicable disease, the Greeks were far behind the Hebrews, and Hippocrates, combating the plague at Athens by building fires fed with aromatic essences in the streets, compares none too well with the author of the Jewish Priestly Code. In another field of health, the promotion of personal hygiene,

* Read April 1, 1931.

the Greeks attained unique preëminence. Nowhere and at no time, not even among ourselves today, has the ideal of abounding physical health and strength and beauty been so fully recognized as in ancient Greece, and nowhere, perhaps, has it been more generally and intelligently promoted. As Sudhoff says, with the "central endeavor of Hellenism, physical invigoration by daily gymnastic exercise, the rest of personal hygiene was in great measure associated, viz., care of the skin by washing and bathing, by swimming and massage; physical cleanliness, including care of the hair and clothing; as well as regulation of diet, rest and sleep, and of the sexual life."

The Romans, too, had their special community health program, associated as characteristically with municipal administration as was that of the Greeks with individual human personality. Their aqueducts, spanning the valleys of Europe on their graceful arches, are still among the most remarkable relics of ancient Rome. The development of sewerage and paving and of regulations as to housing and municipal cleaning were equally notable achievements.

With the advent of the Dark Ages the ideals of personal hygiene and of municipal sanitation were forgotten, so far as Europe was concerned. The spirit of the time was well expressed by St. Jerome in his famous phrase, "Does your skin roughen without baths? Who is once washed in Christ needs not to wash again." Neglect of the material world brought its inevitable retribution in great epidemics which swept over Europe from the East; and such disasters called forth response in the form of a revival of the practices of isolation and quarantine, much along the lines followed in the Mosaic code. From the time of the Council of Lyons in 583 A.D. the movements of lepers were regulated, and from the fourteenth century onward quarantine was practised against the plague; but these were almost the only manifestations of organized preventive medicine from the fall of the Roman Empire down to the middle of the nineteenth century.

The Great Sanitary Awakening. The present-day public health movement, which we hope will be more lasting, and which we know to be more fruitful, than its predecessors, began a little less than a century ago with the publication of a report of the Poor Law Commissioners to the British Parliament, entitled the "Sanitary Condition of the Laboring Population of Great Britain." As in so many instances, the initiative came from a layman, Edwin Chadwick, Secretary of the Poor Law Commission, who was impelled

by a growing realization of the part played by preventable disease as a factor in poverty. He engaged Southwood Smith and other physicians to make the first known sanitary surveys, and the conditions of filth under which the poor (and in large measure the rich as well) were living were so revolting that their description led to a world-wide movement for cleanliness which has justly been described as "the great sanitary awakening." As Sir John Simon saw it, the root of the matter lay in lack of "any high standard of sensibility to dirt. Against accumulated obvious masses of filth, against extreme atrocities of stench, local protests, no doubt, are pretty commonly to be heard, and, at moments when there is panic about disease, may often rise to considerable warmth of indignation, but in regard of the less riotous forms of uncleanliness far too much insensibility is widely shown."

Much the same conclusions were drawn by Stephen Smith and his colleagues in their pioneer American survey of New York City in 1865, when the investigators in a two weeks' canvass found over 1200 cases of smallpox and 2000 cases of typhus fever. In a single tenement house on Mulberry Street, with a transient population of about 320 persons, there had been 240 cases of fever and 60 deaths in a period of four years.

It must be remembered, of course, that Chadwick and Stephen Smith and their associates had no accurate knowledge of the exact measures which should be taken to cope with such evils. They did recognize that certain diseases spread from country to country by a process of infection and that the extent of their prevalence varied in a given locality with the filth conditions which prevailed there. That cholera or typhoid fever was contagious in the same sense that smallpox was contagious, they strenuously denied. In general they believed with Richard Mead a century and a half before, that "The Blood in all Malignant Fevers, especially Pestilential ones, at the latter end of the Disease, does like fermenting Liquors throw off a great Quantity of active Particles upon the several Glands of the Body, particularly upon those of the Mouth and Skin, from which the Secretions are naturally the most constant and large. These, in Pestilential Cases, although the Air be in a right state, will generally infect those, who are very near to the sick Person; otherwise are soon dispersed and lost: But when in an evil Disposition of This they meet with the subtle Parts, its Corruption has generated, by uniting with them they become much more active

and powerful, and likewise more durable and lasting, so as to form an Infectious Matter capable of conveying the Mischief to a great Distance from the diseased Body, out of which it was produced." Thus, "a corrupted State of Air" was "without doubt necessary to give these Contagious Atoms their full Force."

The leaders of the Great Sanitary Awakening by clearing away filth and improving water supplies did, indeed, accomplish measurable results in the control of the more farreaching epidemics of cholera and plague and typhus fever which had devastated Europe since medieval times. Their achievements were seriously limited, however, by the limitations of their knowledge; and fifty years ago the communicable diseases played a rôle which it is now shocking to contemplate. Thus, in the city of New Haven the leading causes of death in 1880 with the rate per 100,000 which they caused were as follows:

1. Pulmonary tuberculosis	262
2. Diphtheria	116
3. Infant diarrhea	98
4. Pneumonia	98
5. Heart disease	82
6. Violence	62
7. Cancer	59
8. Typhoid fever	51
9. Nephritis	49

At this time, however, almost exactly half a century ago, came the great discoveries of Pasteur and Koch which gave us at last the basis for a really effective control of the communicable diseases. By 1880 Pasteur had established the germ theory of disease (1865–1870), Koch had demonstrated the etiology of anthrax (1876) and the gonococcus and the leprosy bacillus had been described (1879). Then began a flood of great discoveries which burst upon the world as Harvey Cushing has said "like corn popping in a pan." Consider the record of one marvellous decade: in 1880, discovery of the germs of typhoid and pneumonia and the pathogenic streptococci and staphylococci; in 1881, the germ of malaria and vaccination against splenic fever; in 1882 the germs of tuberculosis and glanders; in 1883, those of cholera and diphtheria; in 1884, tetanus; in 1885, the cultivation of the gonococcus; in 1887, the etiological agents of meningitis and Malta fever; in 1888, diphtheria toxin and typhoid vaccine; in 1889, the Pfeiffer bacillus and diphtheria antitoxin.

Truly this was the golden age of bacteriology; and during the quarter century between 1880 and 1905, our program for the control

of the acute communicable diseases was placed upon a broad and firm foundation. Bacteriological tests made environmental sanitation, and the purification of water and milk supplies accurate and precise. The insect-borne diseases yielded up their secrets. Laboratory diagnosis of cases and carriers substituted purposeful isolation for gunshot quarantine. Immunology gave us control of specific immunity against typhoid fever and diphtheria. As Pasteur had said when announcing his basic discovery of the etiology of chicken cholera, "How many mysteries in the history of contagions will one day be solved as simply as this."

The New Public Health. By 1905, then, bacteriology and sanitation had placed at our disposal defenses of proved and demonstrated efficacy against the epidemic diseases (with the important exception of influenza which still remains unconquered). Cholera, plague and typhus fever had disappeared from civilized communities, and we had in our hands the weapons to eliminate typhoid, diphtheria and infant diarrhea.

Tuberculosis, however, still remained as in 1880 the "captain of the hosts of death." The city of Philadelphia has the honor of making the first step toward a formal organization for combating this disease through the founding by Dr. Flick of the pioneer Society for the Prevention of Tuberculosis in 1892. In 1894, Biggs, in New York, launched the first official campaign in the world for the administrative control of this disease. It was only in the beginning of the present century, however, that the movement took on national proportions. A second state society was organized in Ohio in 1901, a city committee in New York in 1902 and the National Tuberculosis Association in 1904.

Biggs' early program in the nineties was conceived chiefly on bacteriological lines and was, at first, primarily concerned with laboratory diagnosis, reporting and segregation of individuals who might be sources of infection. Twenty-five years ago, however, it had become clear that the control of tuberculosis depended quite as much on physiology and personal hygiene as on bacteriology and sanitation. It was apparent that in this disease infection was so widespread that isolation could only yield partial and incomplete results, while, on the other hand, the upbuilding of vital resistance was a weapon of almost unlimited possibilities. There had been right on both sides when your College on January 12, 1894, debated the question of notification of tuberculosis. On this occasion,

Flick championed compulsory reporting and DaCosta and Owen Wister opposed it, succeeding in passing a resolution "That the College of Physicians believes that the attempt to register consumptives and to treat them as the subjects of contagious disease would be adding hardship to the lives of these unfortunates, stamping them as the outcasts of society. In view of the chronic nature of this malady it could not lead to any measure of real value not otherwise obtainable." Wister was in error in maintaining that tuberculosis was a hereditary disease, but DaCosta was right in emphasizing that its contagiousness was of relatively low degree.

After ten years of experience, notification of tuberculosis had proved its value, but it was evident that much more than detection and isolation of open cases was needed for the control of this disease. It is notable that the basic elements in the program of the committee established by the Charity Organization Society of New York in 1902 were "research into the social, as distinct from the medical, aspects of tuberculosis," education, development of sanatorium treatment and social relief.

The public health program of the nineteenth century was concerned with the environment of man, with water and sewage and insect carriers, and with the control of the spread of widespread community infections from one person to another. The new public health, initiated by the pioneers of the tuberculosis movement, is concerned chiefly with the individual and with the upbuilding in that individual of the highest degree of vital resistance by the application of the principles of personal hygiene.

With the dangers which lie outside us the community could deal directly by legislation and the exercise of the police power. With the problems of personal hygiene it can deal only indirectly through the medium of education, for our leaders in public health have learned the lesson which other reformers seem slow to master that changes in habits of daily living come by education and not by legislation.

The modern community health program has, therefore, become largely an educational movement designed in an organized fashion to bring the lessons of modern medical science directly into the individual home and to the individual citizen. It is for this reason that the public health nurse has become a central figure in our movement as not only a minister of healing but a messenger of health.

There has been another, even more important corollary of this new conception of public health. It has brought the physician into the community program as its central figure. In the old days, when the health program was concerned with environmental sanitation and the control of epidemics, this was not the case. The health officer might or might not be a physician; but the problems with which he dealt were engineering and bacteriological problems, not medical problems. Today this is all changed. The basis of our community health program is education in personal hygiene, and education in personal hygiene is a personal and individual matter. There are certain generalities as to food, fresh air, exercise and rest which we may teach to all; but the important thing is what food and exercise and rest John Smith needs with his particular constitution and his particular handicaps. If he has tuberculosis, he must have more rest and less exercise than if he has not. The whole program of education for him depends on medical diagnosis at an early stage in the evolution of his disease.

Thus a cornerstone of the development of the community tuberculosis program has been the establishment of diagnostic clinics, not solely or even primarily for the detection of sources of infection who may be dangerous to others, but chiefly for the early diagnosis of a pathological condition, so that the individual concerned may have the advantages of medical advice. Widespread means of propaganda have been developed to bring the individual under the care of a physician. Nurses seek out contacts and those who are physically in any way below par. Today a campaign is under way for the tuberculin-testing and roentgen raying of all schoolchildren as a routine measure, so that tuberculosis can be discovered in its earliest and most responsive stages.

Now this is a new thing in the world, this organized program for a truly preventive medicine. It is by no means confined, however, to tuberculosis. The campaign for the reduction of infant mortality began as a movement for clean milk, an environmental problem. When the first American Conference for the Prevention of Infant Mortality was held in New Haven, in 1909, however, speaker after speaker announced his discovery that success depended not on the number of quarts of milk distributed but on the number of mothers educated to nurse their babies. Today the "milk station" has become a "well-baby conference" whose objective is to bring infants under medical supervision for the teaching of hygiene and the detec-

tion of such deviations from the normal as call for special medical advice. We are recognizing that the pressing challenge of our high maternal mortality can be met only by similar machinery in the form of prenatal conferences.

Since the child is in a special sense a ward of the state it is natural that the new public health should have made rapid progress in the field of school hygiene. When the first school doctors in this country were appointed in Boston, in 1894, they were sent into the schools for a definite police function, to detect and exclude children suffering from communicable disease who might be a menace to others. Today the major interest of the school physician is not in communicable disease at all, but in the detection of incipient physical defects of sight and hearing, diseased teeth, tonsils and adenoids, faulty posture and poor nutrition—conditions which handicap the individual child itself in the enjoyment and efficiency of its daily life.

We shall not comprehend the public health program of the present day if we do not recognize that there is implicit in it a threefold ideal—the safeguarding of the community against environmental causes of disease and the spread of acute epidemic infections, the education of the individual in the principles of personal hygiene and the insuring for that individual of such contacts with the medical profession as shall facilitate the early diagnosis and preventive treatment of disease.

The Community Health Program. Along such lines as these there has grown up during the past ten years a program of community health organization which has become crystallized in a very definite form. This program is based largely on surveys conducted during the past ten years by the Committee on Administrative Practice of the American Public Health Association, and represents not an abstract ideal evolved in the closet but a concrete picture of the best practice in some 300 cities of the country whose actual performance has been studied. In general, the standards set for each particular activity represent the practice of the upper third of the cities in respect to the type of work in question.

The program as worked out in the volume on Community Health Organization prepared for the Committee by Professor Ira V. Hiscock provides for eleven different functions or branches of work which may be summarized very briefly as follows.

There are, first of all, five fundamental activities which represent the older and more traditional aspects of the subject and which are

nearly always the function of the official department of health. These include:

1. Sanitation, including the handling of complaints and the systematic inspection of streets, yards, lots, tenements, public buildings, etc., for the control of nuisances and particularly of such environmental conditions as may be prejudicial to health. Privy vaults and cesspools, care of stable manure, mosquito control and rat control are the most important problems so far as health is concerned.

2. Control of milk and other foods, including licensing of all milk dealers, and of all places where other foods are handled (except in packages) inspection of milk farms, creameries, milk stations and pasteurizing plants, of hotels, restaurants and food stores of all kinds. Effective pasteurization of all milk is the most essential desideratum in this field.

3. Control of acute communicable diseases by securing reporting, by aiding in clinical and laboratory diagnosis, by isolation of cases and carriers (including hospitalization where necessary), quarantine of contacts, distribution of sera and vaccines and special epidemiological studies.

4. Laboratory service, including examinations of diphtheria, tuberculosis, venereal disease, typhoid, etc., for diagnosis and release; chemical and bacteriological examination of water and milk and the like.

5. Vital statistics, including the collection and analysis of reports of births, deaths and cases of communicable disease, with a view to wise planning of the public health program and measurement of its results.

In addition to these five types of community health service which derive from the sanitation and bacteriology of the nineteenth century, there are six others which represent the tendencies of the newer public health. These latter activities are essential to the community health program as a whole, but they need not necessarily be carried out by the official health department. As will be pointed out in later paragraphs, many of these functions may more profitably be performed, in a given case, by voluntary health agencies, by polyclinics or by the private physicians in their offices. These newer activities may be summarized as follows:

6. Tuberculosis control, including reporting and registration

(which must of course be official), diagnostic clinic and consultation service, sanatorium treatment and follow-up of arrested cases, systematic machinery for securing examination of suspects and contacts and provision of open-air rooms, day camps and preventoria.

7. Venereal disease service, covering registration, diagnosis, treatment clinics and follow-up of sources of infection.

8. Maternity and infancy work, involving prenatal care, obstetrical care (including supervision of midwives) and supervision of the health of infants and children of preschool age by means of conferences and home follow-up.

9. School health work, which involves sanitation of school buildings, adequate facilities for physical training, daily classroom inspection for emergency conditions, complete medical examination three times during school life, follow-up for the correction of physical defects and education in the principles of healthy living.

10. Popular health instruction, including preparation of bulletins, newspaper articles, exhibits, motion pictures and radio talks to keep the general public informed as to the bases of disease control and health promotion.

11. Public health nursing. The public health nurse plays a major rôle in many of the specific activities listed above. She is the ideal quarantine officer, since education of the family is essential to effective home isolation of communicable disease. She is the chief factor in bringing tuberculosis cases and contacts to the physician for examination and in maintaining standards of home treatment and follow-up of arrested cases. She is the effective agent in making the home contacts which are essential to the success of work for maternity and infancy and school hygiene. Experience has shown, however, that public health nursing is fully effective only when it is organized on a generalized district basis under competent nursing supervision, except for school nursing which may sometimes be wisely organized on a specialized basis. The ideal program calls for one district nurse for each 2000 people in the general population, rendering bedside care in the home and dealing with all types of public health problems, and this generalized staff should be provided with specialized supervision in the fields of maternity and infancy, tuberculosis, nutrition, and mental hygiene.

We know, too, quite closely what such a community program as

that outlined should cost. In round numbers, Professor Hiscock's plan calls for the following budget, including of course all official and voluntary participating agencies and not the health department alone.

General administration	14	cents	per capita	per annum
Sanitation	10	"	"	"
Milk and food control	11	"	"	"
Communicable disease control (including hospitalization)	29	"	"	"
Laboratory service	10	"	"	"
Vital statistics	5	"	"	"
Tuberculosis control	8	"	"	"
Venereal disease control	9	"	"	"
Hygiene of maternity and infancy	11	"	"	"
School hygiene	23	"	"	"
Popular health instruction	4	"	"	"
Public health nursing	86	"	"	"
Total	220	"	"	"

Thus, a reasonable complete public health program in accord with modern standards costs about $2.20 per person per year.

It has been found possible not only to outline a standard community health program but also to devise a measuring rod for estimating the quantity (and in some respects, the quality) of the service rendered. On the basis of its surveys, The Committee on Administrative Practice has prepared an *Appraisal Form for City Health Work* which is generally accepted as giving a clear and reasonably accurate picture of actual accomplishment. It includes 168 different objective measures of performance which are so weighted as to give a percentage score for each of 14 different types of health activity (including the first 10 of those listed above, with maternity, infancy and preschool work separately tabulated, with nursing included in the various functional activities to which it is related and with the addition of programs for control of heart disease and cancer). The standard of performance is based in general on the practice of the upper third of American cities with respect to the activity in question. To take tuberculosis as an example, a perfect appraisal score requires the following achievements: reporting of 2 new cases each year per annual death, registration of 5 known living cases per annual death; systematic reporting of discharged cases by sanatoria

to the city, 15 clinic visits per annual death, 3 clinic visits per patient registered, 25 per cent of all registered clinic cases in incipient stage, 3 contacts examined for each new positive case recorded at clinic, 50 public health nursing visits on behalf of tuberculosis cases per annual death, 8 such visits per year per case registered with the nursing service, 10 nursing visits to postsanatorium cases per annual death, 250 patient-days of institutional care per annual death, 25 per cent of hospital and sanatorium admissions in incipient stage, 10 per cent of such admissions under twelve years of age, 1 per cent of grade-school population in open-air classrooms, day camps or preventoria.

Such formulæ no doubt sound artificial and arbitrary, and in particular instances they give an unfair picture of a local situation. They have, however, been derived from careful study of actual performance, checked and rechecked in hundreds of surveys, and in actual use they serve in general to pick out the strong and the weak points in a given community program.

Thus, in the recent survey of Philadelphia by Dr. Haven Emerson, it was shown that communicable disease control and maternity and infant hygiene reached a very high standard, scoring between 88 and 98 per cent; that milk and food control, laboratory service, vital statistics, tuberculosis control and popular health instruction were of fair grade, scoring between 71 and 82 per cent; while marked deficiencies appeared in school hygiene (60 per cent), preschool hygiene (52 per cent) and sanitation (41 per cent). The low score for school health work is due to a defective program in the parochial schools, lack of attendance of parents at examinations and inadequate facilities for correcting defects. Dr. Emerson, however, finds the quality of such work as is performed to be very high and refers to "the excellences of the work of the school doctors and of the nursing work and the high-grade direction of this division." The low score for preschool work is due to the very small development in this field and the low score in sanitation chiefly to the quality of the public water supply.

Problems of the Future. The public health program as it has developed during the past half-century has achieved results which are nothing less than astounding, and these results have altered the whole fundamental picture with which the public health program of the future must deal. I have quoted in an earlier paragraph the

death-rates for the city of New Haven fifty years ago. Let me cite them again, with the record of 1930 for comparison:

DEATH-RATES FROM CERTAIN PRINCIPAL CAUSES IN NEW HAVEN, CONN.
(Including all Causes which in Either Year Caused Over 50 Deaths Per 100,000 Population)

Cause of death.	Deaths per 100,000	
	1880	1930
Pulmonary tuberculosis	262	36
Diphtheria	116	1
Infant diarrhea	98	2
Pneumonia	98	100
Heart disease	82	231
External causes	62	59
Cancer	59	124
Typhoid fever	51	0
Nephritis	49	58
Cerebral hemorrhage	*	116

* Not tabulated in 1880.

The death-rate from pulmonary tuberculosis has dropped to less than one-seventh of the rate of fifty years ago and this cause of death has fallen from first place to seventh. Diphtheria, infant diarrhea and typhoid, ranking second, third and eighth in 1880, have practically disappeared in 1930. Pneumonia, external causes and nephritis show little change, while heart disease, cerebral hemorrhage and cancer exhibit enormous increases.

The problems of the future are obviously pneumonia, cancer and the diseases of the heart and arteries. It is here that further gains must be made if they are to be made at all, and there is no reason to despair of progress in this field.

It has been said that cancer is as controllable as tuberculosis, which is at present untrue of the disease as a whole. It is true of cancer in certain sites, however, and the method of approach is an essentially similar one. Education of the public to recognize early signs and consult a physician, prompt diagnosis and immediate treatment are as valuable in many forms of cancer as in tuberculosis —the only difference being that "treatment" involves an operating room and not a sanatorium. In the case of heart disease, we may look for progress from the control of such underlying factors as focal infections, rheumatic fever and venereal disease; and, whatever the underlying cause, prompt diagnosis and appropriate hygienic care will postpone serious results and in youth may lead to complete cure.

In recognition of these facts, the last edition of the *Appraisal Form for City Health Work* includes special sections devoted to heart disease and cancer as problems which must be included in a complete community health program. Definite standards are set for continuing statistical surveys, for diagnostic clinic service, for hospitalization and nursing follow-up, very similar to those established for tuberculosis and venereal disease.

Finally, behind all these more familiar and obvious problems lies the vast and almost untouched field of mental and emotional maladjustment. We know that the number of institutional beds provided in a given community for extreme mental diseases and defects is approximately equal to the sum of hospital beds for all other diseases and defects taken together, and we believe that this ratio fully holds for the less acute types of disorders. It is estimated that 1,000,000 children and young people now in school and college are destined to be inmates of mental hospitals at some time in their lives. In an average group of four-year-old children in a New England city, according to Emerson, over one-third were found to have "characteristic behavior problems that are likely to develop into personal or family embarrassments." Yale University has four full-time psychiatrists who find themselves fully occupied in caring for 5000 normal young men. The handicap which most of us suffer from fears, jealousies, suspicions, resentments, insecurities, hesitations, which have their origins in the subconscious level are beyond estimation.

The lack of general awareness of these problems and, above all, the lack of properly trained professional personnel, must make progress very slow in this field. We must look forward, however, in the future to an extension of facilities for custodial care of mental diseases and defects, to the establishment of psychopathic wards in general hospitals for "first aid to the mentally injured" and to marked expansion of psychiatric, mental hygiene, child guidance and behavior clinics to make the work fundamentally preventive and truly efficacious.

Voluntary Health Agencies. For the sake of clarity we have considered the various aspects of the community health program as if they formed parts of a single consistent whole. It must be clearly recognized, however, that it is in no sense intended that all the activities discussed should be carried on by the official health department. The health officer of any governmental unit must be

recognized as the one person officially responsible to the community for the health of its people. It is his task to formulate general policies of health promotion and to secure coördination of all health agencies within his jurisdiction. In large communities he should be provided with specialists competent to direct the entire community program in such fields as child hygiene and the control of tuberculosis and venereal disease. Each particular function in the field of health promotion should, however, be discharged by the particular agency best fitted to perform it. That agency in many instances may be the private practitioner of medicine or the voluntary health agency.

The subcommittee of President Hoover's White House Conference on Child Health and Protection, dealing with the Relation of Official and Nonofficial Agencies in Public Health, estimated that about $100,000,000 a year are spent in the United States for health promotion and that more than one-quarter of this sum comes from various nonofficial agencies, foundations, national associations, life insurance companies and local child welfare, tuberculosis and nursing groups. Between 40 and 50 per cent of all the public health nursing service and about one-fifth of all public health clinic service is provided by voluntary agencies.

The Rôle of the Medical Practitioner in the Community Health Program. Finally, we come to the problem of the rôle of the practitioner of medicine in this complex and diversified community health program. The sharp separation between official prophylaxis on the one side and private therapeusis on the other which was tenable in 1900 is no longer tenable today. The health officer realizes that tuberculosis and venereal disease and heart disease and cancer can be controlled only by medical treatment; and the private practitioner in his office desires to become a hygienic advisor and not merely a last resort for the repair of a machine which has already broken down. The fields of public health and private practice have begun to overlap, and the first result has in many cases been a certain measure of mutual tension.

That there is no real and fundamental conflict involved seems basically clear. The first stages of the public health movement which were concerned with environmental sanitation really did damage the selfish interests of the medical profession and in a serious degree. The purification of water supplies cut off a substantial source of income to the doctors of your city and in many southern communities antimalarial campaigns have well-nigh wrecked local medical

practice. Yet against these advances the profession never raised a murmur of dissent.

The new public health does not decrease but greatly increases the total volume of medical practice. Its whole emphasis is on early diagnosis and prompt medical treatment. It hopes to reduce the burden of acute disease, but the number of hours of medical service needed for dealing successfully with tuberculosis and heart disease and cancer in a preventive fashion will not be less but more than the number of hours employed in the past in attempts to repair damage already accomplished.

There can be no just quarrel between the profession as a whole and the leaders in the public health program. There may, however, be conflict between those members of the profession who are within and those who are without the field of organized preventive service. Our great problem, perhaps, is to determine what part of the preventive program can be carried by the private practitioner in his office and what part must be provided in an organized form by official and voluntary health agencies, including hospitals and dispensaries. In discussing this general problem five years ago, I said: "The health officer as an official responsible to the community for the promotion of the public health in all its possible aspects should consider it his primary responsibility to work out in coöperation with the medical profession a wise solution of this problem, a solution which will preserve and maintain all that is best in the noble traditions of the healing art. In the last analysis it will be the duty of the health officer of the future to see that the people under his charge, in city or country, in palace or tenement, have the opportunity of receiving such service as that outlined above and on terms which make it economically and psychologically easy of attainment, and himself to furnish such service if and when it cannot be provided in other ways."

The resources of modern preventive medicine must be made available for all the people; but the wise health officer is the one who does nothing himself that he can get the private practitioner to do for him. In such cities as Minneapolis the health supervision of infants has been so well developed by pediatricians that public child-welfare conferences are open only to the indigent. In rural areas prenatal and venereal disease service can only be rendered effectively by the private physician in his office, and we have had notable success in rural Connecticut through subsidizing rural

practitioners to render service of this kind. In Detroit a particularly important experiment has been made in the direction of transferring diphtheria immunization from the public clinic to the doctor's office. In November, 1928, the actual administration of toxin-antitoxin was turned over to the private physicians under a definite agreement with the County Medical Society. Nine hundred of the 1500 physicians of the city have agreed to do immunizing and Schick testing at a fixed rate, the health department to pay for all patients who cannot afford to pay themselves. The number of children immunized has fallen somewhat as compared with previous years when public clinics were operated and the expense to the health department is greater than under the clinic plan. Dr. Vaughan, the health commissioner, believes, however, that the greater convenience to the public, the higher proportion of preschool children immunized and, above all, the establishment of new contacts with the private physician as a preventive agent more than compensate for any sacrifices involved.

Looking at the whole problem broadly and objectively, it seems to the writer that the essential issues may be stated as follows:

Modern medical science has vast potential resources for the good of humanity along lines of preventive service. These resources are not today being fully utilized. Much even of acute and chronic illness is uncared for. Fundamental prevention is ignored. Vast sums are wasted on quacks and nostrums. In a recent survey made by the Committee on the Costs of Medical Care in a rural county of Indiana it was found that 34 per cent of all moneys expended for health were spent for drugs and medicines, 29 per cent for medical service and only 1 per cent for public health. A similar study in your city shows that Philadelphia is better off. The largest slice of the health budget goes, as it should, to physicians (26 per cent); but even here drugs and medicines, most of them useless and many of them harmful, absorb 20 per cent of the total, while public health gets less than 2 per cent.

There is a woeful gap between supply and demand which can only be bridged by salesmanship, and salesmanship is something which the traditions of the medical profession cannot tolerate on the part of those members who might profit by the sale. The health officer, however, is personally disinterested in this matter. He can and should, as I believe, be as active in warning against the dangers of quackery and self-medication as in warning against the dangers

of polluted water; as active in promoting the use of good medical service as in increasing the consumption of milk. The basic ideal of the new public health is that which Dr. Olin West has described as "the one great outstanding problem before the medical profession today—that involved in the delivery of adequate scientific medical service to all the people, rich and poor, at a cost which can be reasonably met by them in their respective stations in life."

DISCUSSION

Dr. E. L. Bauer: I am quite impressed with Dr. Winslow's paper this evening, a most comprehensive presentation and one upon which he should be complimented highly. He has brought out many points, all of which may be criticized in a very complimentary way. I am struck, first, by the chart in which he assigns the expenditures necessary for the many public health activities in a community. So far as private agencies are concerned in Philadelphia, I am not in a position to say, but the annual budget of the Department of Public Health comes to approximately one-sixth of that which is on Dr. Winslow's chart. His chart calls for $4,400,000 for public health work in Philadelphia and that is approximately one-sixth of what Councils appropriates annually. As for the outside agencies, they do help and do a great service in supporting public health work in Philadelphia. If we throw their budget in we might come somewhere near approximating the doctor's estimate. I feel Dr. Winslow is quite right when he says that the Department of Health should in some way have an advisory or directing interest in all community health activities for two reasons: One in maintaining a high grade of work and, second, which he did not mention but which I believe is of equal importance and needs careful attention, is the danger of overlapping activities. The private institution feels that it will reach out here and reach out there and splashes around the pond, hoping the rings will get to the sides in all directions. A multiplicity of rings from one splash here and one splash there means overlapping of rings. The consequence of overlapping activities will result in more money spent for less return than if the Department of Health itself could direct or establish supervision over stations and districts, and so on, as done, for example, through the coöperation of the Babies' Hospital and the Department of Public Health. The fifth and sixth wards are set aside for the Babies Hospital. The Department of Public Health has not interfered with the work, satisfied that the character of work done is excellent. We know from inspections and the reports that we get from the Babies' Hospital of the character of work being done there, and our doctors and nurses stay out of that district entirely. Other coöperative centers are established in points about the city. That type of work must be taken into consideration in the budget for public health work.

New legislation should be cut to a minimum in public health work. No public health officer wants to be a policeman, but sometimes it has to be done. Punitive measures will not be successful in the long run, and the Public Health Department does not wish to engage in police work primarily. We are most interested in the educational angle. Before we go into the educational angle, let me speak of that report of Dr. Emerson's in regard to the water supply in Philadelphia. It created a furore in Philadelphia because many people have a dubious attitude in regard to the water supply. I think Dr. Emerson had in mind the burden on the filtration plants and the fear that they would break down. There is a multiplicity of them; they are scattered and if one breaks down there are others to take up the burden. Chlorination is carried out consistently. I do not think there is sufficient evidence upon which to give us this low grade. I think every body in the Health Department took umbrage at this criticism, especially in view of our low typhoid rate. You will recall Philadelphia in the past was known as a typhoid city; today it has less than 1 death per 100,000 from typhoid. It speaks well for our water supply. As far as our parochial school inspection is concerned, a sincere effort has been made to improve that weak link through the acquisition of a new large group of nurses and school inspectors. How that is going to work out will be seen at a later date. Public health means public education in health habits. That is accomplished in large measure through the schools, the parents' associations, through the Well Baby Center, Prenatal Centers, Health Centers, etc. We make examinations of tuberculosis suspects and contacts at our centers and teach the prevention of this disease. We do recognize as a great force for good the work of Dr. Opie and his examination of school children through the medium of *x*-ray and the tuberculin reaction. Some of this work is in the preschool-age group, and it is our idea, as practised at Jefferson, and the idea of Dr. Charles Hendee Smith, at Bellevue, that the preschool age is one that should be approached. We should cover the school child, no doubt, but if we want to get anywhere we should go to that neglected preschool child. We have educated the mother to come to the center, the hospital clinic or to the physician for prenatal care. We can hold on to her as long as her child is on the breast or on a formula. We *must* hold on to her and her child until it is ready for school, because in that interval, as soon as the child gets on the ground and runs around, it is forgotten until it has unfortunately passed some danger signal or has developed some disease or deformity, to be picked up in school later on with some condition that should have been avoided or corrected in that preschool age. The clinics have shown their worth in the preschool age as far as the actual results of the work are available. The chart there shows us the New Haven tuberculosis rate. I have here the death-rate per 100,000 in Philadelphia in 1912. Our rate for 100,000 was 190; in 1930 it was 81.38. One of the functions of our centers is the prevention of definite diseases. Diphtheria is one of them. In 1900 Philadelphia had 5008 cases and 1042 deaths, a death-rate of 80.4 per 100,000. In 1925 there were 3887 cases and 315 deaths, a rate of 16.1 per 100,000. In 1930 there were 635 cases and 48 deaths, reducing the

mortality-rate to 2.45 per 100,000. This reduction has been due to the intensive drive against diphtheria by means of toxin-antitoxin in our health centers. Some cities depend entirely upon the physician to immunize against diphtheria and smallpox. Dr. Vaughan introduced this latter method in Detroit; Dr. Wynne has introduced it in New York City. The White House Conference shows that New York and Detroit have a rather high immunization record in the preschool-age group, and Philadelphia has a standard of 28 per cent in this classification. When Dr. Hamill instituted that investigation in Philadelphia he carefully sought out the groups that had not been met in hospitals and centers under our control, so that his percentage represents to my mind what the doctor is doing in private practice. Now I have in my possession, signed and sealed, the name and address, the age, the parent's signature, the signature of my inspector who performed the work, the immunization record of 53 per cent of the children in Philadelphia born between 1925 to 1930. They are there as receipts, as evidence, that the work of immunization has been performed by the Department of Public Health, as well as permission to perform the work. This bears out my contention that the 28 per cent was either a very superficially obtained figure, or is more an evidence of what the physician is apt to do in communities where the Department has no access. It shows that it is a matter for the public health officer to drive and drive to get this sort of work done. We are through with smallpox. So far as we are concerned it is a historical disease. We only see a couple of cases in three or four years in Philadelphia, and they come from outside the city. There have been no smallpox deaths in Philadelphia since 1925. Not only is the school population vaccinated, but upon exposure large blocks of the population are vaccinated. We rope off areas of three or four blocks, go in and vaccinate 3000 or 4000, stamping out smallpox by the only possible way, vaccination.

I just wish to comment favorably upon mental disease clinics. We have established such a clinic at Jefferson, unfortunately misnamed, and a splendid psychiatrist is attempting to do all that he can with the children of our community, both old and young. Again, let me compliment Dr. Winslow upon his excellent presentation. Thank you.

Dr. Randle C. Rosenberger: We all agree with Dr. Bauer's remarks regarding immunization of children against diphtheria. I have a compilation of the last ten years of water supply and typhoid-rate, and in the last three years the death-rate from typhoid is less than 1 per cent. I am very much surprised that our water was condemned as it was, and, considering our source and the type of water we have to work with, together with the extreme burden placed on filtration plants, I think we have a very good water supply. I have made examinations for many years of institutions, private homes and swimming pools, and I have yet to find sewage pollution in the last five or six years. Of course, this paper is so extensive that it is impossible to criticize fully because everything has been brought out so wisely by Dr. Winslow that it really does not need criticism but emphasis and reiteration.

The prevention of disease, the attainment and promotion of health are the objects we are striving for in Public Health.

Avenues through which problems may be pursued are many and variable and the education of the general public is the goal for which we are working; whether this be by using the daily paper, circularizing the home, the establisment of clinics, or informal gatherings concerning public welfare.

If we could awaken the public to a realization of the benefit of an early diagnosis, and consulting the doctor upon the first indication of illness either in the infant or grown-up, a great many serious diseases and results might be avoided.

In industries it is well known that the workers must report at once upon the inception of the most trivial injury and thus severe and fatal infections are oftentimes prevented. True it is, that some workers are reluctant to take proper precautions as the wearing of goggles or respirators or the selection of proper tools and as a result serious accidents and loss of life have occurred.

In ordinary walks of life despite suggestions made by the physician or health officer, precautions or warnings are not generally heeded at the time and consequently disease may have made marked headway.

We are all agreed that one of our biggest health problems before the establishment of the prenatal clinic was the heavy mortality of infants and potential mothers. Now, many mothers' lives have been saved and the birth of healthy living children has been greatly augmented. Then follows the proper care of the infant regarding diet, bathing and sunshine. From this tender stage it is carefully watched and groomed, and the beginning of protective measures introduced in the form of vaccination against smallpox. The preschool age is the really precarious one when communicable diseases stalk abroad: we now protect it against diphtheria by toxin-antitoxin or anatoxin. The slightest indisposition of the child should be immediately looked into and isolation, where indicated, should be practised at once. Although it is claimed that isolation has not really cut down the incidence of communicable disease, yet it still serves as one of our most formidable procedures against the dissemination of disease.

The health officer may be, and is handicapped by politics in one respect in employing nonefficient assistants or in another—the ignoring of sanitary regulations passed by the legislature. In this respect we also may have handicaps by reason of apathy or indifference or both in certain practitioners who fail to fall in line for the common good.

In all the problems of public health we have failed to see where any practitioners have had marked curtailment of every-day practice, despite the almost obliteration of certain diseases that had a very high incidence a few years ago.

While the communicable diseases appear as the most important in the preschool age, the consideration of tuberculosis and venereal diseases must never be lost sight of and we must keep everlastingly after them.

Just how intensive a campaign against venereal diseases should be is a matter for careful consideration, but we doubt if it will ever get to the general point of the authorization of the health officer to demand examination of any person who has or is reasonably suspected of having venereal disease.

A very able factor in dispensing knowledge of cleanliness and the dangers of miscellaneous infections is the teacher in the elementary schools. I know of many instances where the teacher has had a great deal to do with pupils and for the pupils to pass this knowledge on to the parents with markedly changed methods of living and looking differently upon simple things as brushing the teeth, keeping the body clean, and also as to proper or more assimilable foods.

The rôle of the social worker we are all familiar with as a faithful, loyal messenger of health.

With all of these problems mostly devolving upon the future of the child, yet we must not forget the problems that now particularly menace us in the form of cancer and diseases of the vascular system.

Finally, health being attained, it must be maintained by pure water, pure, safe milk and the proper disposal of all wastes, especially sewage.

Dr. Frank A. Craig: The point that seems to strike one in listening to any discussion that has to do with public health today, the most striking thing is the practical basis upon which facts are founded. Formerly one would hear in talks upon public health such remarks as "Do you think we had better do this?" or "Perhaps we should do that." Today this uncertainty is absent, as the public health physicians have brought our knowledge to the point where they can outline a program which may definitely be placed in the hands of any community. This program has been so perfected that it is no longer a question of what the doctors think but what the public is willing to spend. On this chart you can see it set out in dollars and cents just how much each branch of public-health work will cost. It is a remarkable fact that it has been so worked out that they can present a program which will show just how much disease prevention they can provide for a certain price. The public can have it if they are willing to pay for it.

That portion of the program relating to tuberculosis is the one in which I am most interested, and while you will notice on the chart that the death-rate from this disease has shown a decided reduction in New Haven, this has not reached a point where it is comparable with the reduction in typhoid fever or diphtheria. I regret that Dr. Winslow, in speaking on the method of dealing with tuberculosis, rather minimized the value of isolation of the patient. I know that in all the preventive work of the National Tuberculosis Association at the present time emphasis is laid upon the building-up of the health of the individual to prevent the development of disease in those already infected. I am still of the opinion that we would make greater progress against tuberculosis if we would make a greater effort to secure isolation of the infectious patient for as long a period as possible. I took from Dr. Winslow's remarks that he minimized the value of isolation, which I feel is one of the most valuable preventive measures we possess.

This brings to my mind a point recently brought to my attention, when discussing this subject with Dr. Flick, who is very much interested at the present time in having the general hospitals admit tuberculous patients.

We are all interested in having our patients with advanced tuberculosis taken care of near their home. We are also anxious to have these patients isolated from the family as far as possible. It seems to me that Dr. Flick's plan would not only secure these two valuable results, but would also provide the resident physicians and nurses with instruction in the treatment of this disease. We insist upon our medical graduates serving in general hospitals before they are permitted to practice, and insist upon nurses serving in a general hospital before being permitted to practice their profession, in both instances with the object of providing them with the proper experience in the treatment of disease. And yet with possibly one or two exceptions, none of the hospitals of this city admit patients suffering from this still very prevalent disease, and neither the resident nor nurse ever sees a case of tuberculosis during his or her service. It seems to me that this plan would provide a wonderful opportunity to not only secure the desired isolation of our patients, but also to give a greatly needed training to our physicians and nurses in the treatment of this disease.

Dr. Seneca Egbert: As has been intimated, this address of Dr. Winslow's might furnish themes for a number of extensive papers instead of a few short discussions, and anything that may be said must seem fragmentary in comparison.

In developing a community health program the importance of habits as factors in the causation of the diseases of middle and advanced life should not be overlooked, especially the habits that are developed during adolescence and immediately thereafter. This is a cogent reason for periodical medical examinations at this time of life and of stressing their value and importance to young men and women, as was done in the Public Health Day exercises last week when the periodic medical examination was the chief subject brought to the attention of the public.

Although the development of immunity and resistance to disease in the individual must always be largely a matter of private concern and social effort, we have had the demonstration that it can be aided and fostered by official action in the recent successful campaigns in this city for the general inoculation of preschool as well as of older children against diphtheria. Dr. Bauer has given you the figures, which are almost marvelous; they would have seemed miraculous and beyond belief twenty-five years ago. As a supplement to them, I might tell you that last year, 1930, the deaths in this city from diphtheria comprised but two-tenths of 1 per cent of the whole mortality. In fact, these together with those due to scarlet fever, measles, whooping-cough and typhoid fever made less than two-thirds of 1 per cent of that mortality.

On the other hand, and supplementing the figures on Dr. Winslow's chart, the mortality from cancer in this city has risen from 3.38 per cent of the total in 1900 to 10.04 per cent last year; that from nephritis and renal disturbances from 6.53 to 10.79 per cent; that from diseases of the heart and circulatory system from 8.29 to 22.28 per cent. The mortality percentage for pneumonia remains almost unchanged—9.16 and 9.15—and that due to tuberculosis shows an encouraging reduction from 11.49

in 1900 to 6.18 in 1930. It would seem that the direction in which our future efforts should be exerted is made clear to us. The brilliant talk that Dr. Winslow has given us tonight shows why stress should be put upon the prevention of cancer and heart disease and the other degenerative maladies of advanced life, and why we should continually impress upon the public the importance of these affections and the need for regular and thorough physical examinations.

Attention should be directed to the need for the rapid future development of industrial hygiene and medicine, and of stressing the importance of this in the information and education of the public at large. It will be interesting to note hereafter how much the present economic depression has to do with bringing about more interest in and attention to the physical welfare of the workman. The present indications are that industrial hygiene is rapidly becoming one of the foremost as well as one of the most important subdivisions of the art and science of hygiene in general.

Dr. Winslow referred to the value of the public health nurse in bringing the lessons of modern medical science directly to the home and to the individual citizen. We should not fail fully to appreciate the value of her and her sister worker, the public school nurse, as missionaries of sanitation and educators in the broad field of hygiene and preventive medicine. In this connection the statement at a public meeting in this city a few months ago by the head of the public school nurses of this city is worth remembering, viz., that the school nurse was the real teacher of hygiene and sanitation to the coming generation because of the intimacy of her contacts and her direct helpfulness to the children in time of need, and that the effect of her influence and teaching far exceeded that of either the teacher or the school physician.

Lastly, I am pleased that Dr. Winslow outlined for you Dr. Hiscock's ideal budget for a community health program. An annual expenditure of $2.20 *per capita* may seem excessive to some, but we should remember that all of this is not to come out of the public purse, and most of us here can realize the remarkable results that might be attained if such a total fund were to be used in proper ways for even a year or two, if not continually and systematically in the future. I note that the suggested budget allots 11 cents *per capita per annum* for milk and food control, which is less than the cost of a single quart of good milk in the market today. The present average consumption of milk in this city is almost exactly one-third of a quart *per diem*, and the cost of good milk 15 or 16 cents a quart. So the budget allowance for supervising and caring for the safety and wholesomeness of the total food supply for the whole year would be covered by the cost of the average individual's milk supply for only two days.

May I express again our indebtedness to Dr. Winslow for his most interesting and instructive address.

Dr. John M. Fisher: A great deal of attention today is called to the prevention of disease. I notice on the chart that cancer increased in 1930. Dr. Winslow referred to the fact that the disease is becoming more frequent

but said nothing with reference to the prevention of cancer. A great deal has been done in recent years in the prevention of cancer of the uterus. Cancer of the uterus and cancer of the mammary gland, it is well nigh conceded, occur oftener than cancer of all the organs of the body in the male combined. The question is which is the more frequent, cancer of the uterus or cancer of the mammary gland? In from 5 to 10 per cent of cases of this disease in the uterus it is confined to the body of the organ. If there is a favorable location for cancer it usually is in this location, and if the disease is discovered in its incipiency and even in some cases where it has progressed to a considerable extent, a complete hysterectomy often effects a cure. The recurrence of cancer in such cases is not very frequent when operation is done early. Now as to cancer of the cervix, I think the gynecologist today is doing more for the prevention of cancer in his treatment of obstetric traumatisms than any other doctor in the community. As pointed out a number of years ago by Kelly, in thousands of cases he had only encountered 3 carcinomas of the virgin cervix and in 1 of these a dilatation and curetting had been done. Cancer of the virgin cervix is extraordinarily rare and yet cancer of this structure in women who have borne children is comparatively frequent.

Now what has been done for the prevention of cancer of the cervix? Dr. Hunner, twenty-five or twenty-six years ago, introduced the use of the cautery. In cases of cervical lacerations with everted lips and erosions, and where the condition has not resulted in cystic or interstitial hypertrophies, the cautery usually is sufficient to correct the abnormality and thus prevent the formation of cancer. In a recent communication Dr. Hunner states that he has not seen but one case of cancer following the use of the cautery. This was in one of his early cases and he felt confident some mistake had been made in the microscopic diagnosis and that cancer was present when the cautery was used. By the use of the cautery on amputation of the cervix, with the end in view of removing the diseased part of the cervical mucosa, cancer degeneration can be anticipated and prevented.

Dr. Winslow (closing): About some of the problems that have been raised: Dr. Bauer outlined admirably the need for coöperation between the voluntary agencies and the Health Department. I think the possibilities of such relationships have been well illustrated in my own town. Some years ago the Health Department had a clinic for tuberculosis of its own separate from the clinic at the University, and the Health Department had two or three nurses doing a rather inadequate type of work in this field. There was no head to the whole program. The Health Department then put in a full-time Director of Tuberculosis. The first thing he did was to turn over the nursing work to the Visiting Nurse Association and the clinic to the University, but he headed, controlled and directed the whole program. It seems to me that very well illustrates a happy relationship. Dr. Rosenberg is quite correct about the present satisfactory water supply of Philadelphia. There is no doubt at all that the water supply has so far been satisfactorily handled that no typhoid has

resulted from it in recent years. I take it that the only criticism that has been raised is based on the fact that bacteriological analysis indicates that the supply is on the danger line. Trouble has not happened, but it may at any time. The results of the present treatment are a triumph considering the quality of the water. What is indicated, however, as clearly desirable, is a cleaning-up of sources so that such a tremendous burden will not be put upon the filtration plants. As to what Dr. Rosenberg said of venereal diseases: There is a growing conviction that follow-up is likely to be more fruitful in the future than it has been in the past. It seems to many experts that the foci of infection are not so widespread as was once thought. The number of persons having syphilis in a communicable form is not so enormously large. It is quite possible that we may before long consider syphilis not as a social disease, but as a series of local epidemics, and may hope to control it as such. I am glad the doctor brought up the point in regard to tuberculosis—I did not mean to give the impression that control of infection was not important. I agree with all he said, but the actual control of tuberculosis has been largely accomplished by hygienic rather than bacteriological measures. Dr. Craig also spoke about the importance of an approach on financial grounds. I would like to cite a recent survey made in Cattaraugus County, N.Y. The cost of the very complete health program for a population of 70,000 amounted to $150,000 a year. We made a careful study and taking three diseases alone, tuberculosis, diphtheria and infant diarrhea, the decrease was so marked as compared with the previous trend that these decreases amounted to a reduction in mortality equivalent to a saving to that community of $300,000 a year. I was very much pleased that Dr. Egbert brought out the importance of industrial hygiene. Dr. Cornell is, also, of course, quite correct in emphasizing the seriousness of mental hygiene. I should not wonder if we should see in the next twenty years a development along this line that will be surprising. I agree with what he said of the Health Department's function as an agent in postgraduate education. That has happened in Detroit and is certainly one of the most useful functions the Health Department can assume.

Dr. McCrae made a motion, seconded, that a vote of thanks be tendered Dr. Winslow.

RECENT EXPERIMENTAL CONTRIBUTIONS TO SYPHILIS*

By JOSEPH V. KLAUDER, M.D.

In the time available it would be impossible to review all recent investigations in the field of experimental syphilis. General conclusions of important studies only will be given.

One phase of experimental study of syphilis, confined largely to the infection in the rabbit, concerns the question of immunity. From the studies of many investigators there is no satisfactory evidence of a humoral rôle of immunity in syphilis. Attempts to produce active immunity in man or in the lower animals, by injecting filtrates of syphilitic tissue, killed and living syphilitic virus and cultures of Spirochæta pallida have been generally unsuccessful.

Attempts at passive immunization with the serum of immune persons, or with the serum of animals infected with syphilis, or treated with dead culture of Spirochæta pallida have proved fruitless.

There is little status to the immunologic rôle of such antibodies that have been demonstrated in the serum of syphilitic patients or of animals experimentally infected with syphilis. A number of workers have demonstrated agglutinins in the serum of rabbits after injecting them with culture of Spirochæta pallida. In these instances however, no agglutination occurs with virulent organisms. Eberson demonstrated immune bodies in the serum of patients with latent syphilis. Employing the same technic, his results have not been confirmed by Manteufel, Richter and Worms, Koler and Rule, nor by myself. It, therefore, appears that the mechanism in immunity in syphilis is largely cellular.

Heretofore, the conception of immunity against second attacks of syphilis was in accordance with Neisser's view that resistance to reinfection and latency are due to nonreaction of the tissues, or "anergie" as Neisser called it, a condition said to depend entirely on the continued presence of the virus. Conversely, the appearance

* Read May 6, 1931.

of a second chancre is commonly held to be proof that the first infection was eradicated.

This conception has been somewhat altered through the work, notably of Kolle, Brown and Pearce and Chesney. Their studies have shown that the response of a syphilitic rabbit to a second infection with syphilitic virus depends upon whether a heterologous or homologous strain of pallida is used for a second inoculation, and the time at which the inoculation is made. Up to the sixtieth day of the infection in rabbits, a second chancre can usually be produced with homologous strain, but rarely after the ninetieth day. Treatment of the animal modifies this occurrence. If treatment is begun early in the course of the infection, a second inoculation is possible. If treatment is postponed until after the ninetieth day, the animal will not acquire a second chancre when inoculated with homologous strains. In animals thus treated and reinoculated, Chesney and Kemp injected different organs of these animals into other rabbits, with negative results. Considerable controversy has arisen as to whether the animals failed to develop a second infection because of a definite immunity, or by virtue of the fact that they still harbor the infection, as Neisser and also Kolle contend. The crux of the matter is, whether or not it is possible to eliminate syphilitic infection in the rabbit by treatment. Proof of that rests solely upon negative results of inoculation of various organs, notably lymph nodes, into other animals. It must be borne in mind that it is impossible to emulsify an entire rabbit in order to inoculate all of it into other rabbits.

Reinoculation with heterologous strains more than ninety days after the first inoculation is successful in over 44 per cent.

In man, so far as it is known, acquired immunity against a second infection seems to apply to all strains, and there is some evidence, from the studies of Jahnel and Lange, that syphilis in man protects even against yaws.

There is no convincing evidence that there exists strains of Spirochæta pallida having a propensity for attacking different organs of the rabbit. This applies more forcefully to man.

Immunological studies in rabbits have a distinct clinical bearing in relation to reinfection in man, that is, a new infection in a patient presumably cured of a previous infection. The cases of reinfection have become much more numerous since the use of the arsenicals, and especially in persons receiving intense treatment in the early period of infection. These studies suggest that reinfection in man

is no criterion of cure. It is impossible to establish a frank limit between superinfection and reinfection, that is, where the one ends and the other begins.

Truffi, correlating his studies of experimental syphilis in rabbits to its clinical application, states that, while there are no absolute proofs as to when a syphilitic may be regarded as cured, it may be held, from a practical point of view, that syphilis in man is curable.

An interesting phase in the study of immunity in experimental syphilis is the occurrence of symptomless infection. Schlossberger and Kolle showed that in mice inoculated with Spirocha ta pallida or with Spirocha ta pertenuis, the virus spreads through the tissues without provoking any reaction. In these animals, although the blood always failed to infect rabbits, inoculation with lymph nodes and spleen always succeeded. Likewise in rabbits, symptomless infection has been observed. Kolle was able to produce such infection in male rabbits through copulation with infected females. It has been pointed out that the same thing may happen in man, and that such symptomless, untreated human carriers may be an important cause of the spread of the infection.

Brown and Pearce, from extensive studies of the course of syphilis in the rabbit, emphasizes the important rôle of local tissue reaction to the infection in preventing or modifying the development of generalized lesions. Early or late interference with local tissue reaction alters the course of the disease. By experimental means, they could increase or decrease the incidence of involvement of certain organs. The eye and the nervous system were afforded less protection by tissue reaction taking place elsewhere. The occurrence or nonoccurrence of eye lesions was determined by the prior course of the infection. If an animal has had severe secondary bone and skin lesions, eye lesions in such animal were rare, *i. e.*, the immunity was such as to protect the eye from developing lesions. On the other hand, if there were very few bone or skin lesions, eye lesions were constant. These studies correlate clinical observation relative to the incidence of neurosyphilis appearing after insufficient treatment in the early stage of syphilis, and supports Fournier's dictum, that neurosyphilis was infrequent in patients having a diffuse and well pronounced secondary eruption. These studies also correlate certain phases of interstitial keratitis as seen clinically.

Chesney summarizes the status of acquired immunity in syphilis as a state of resistance which evolves slowly, not always complete,

general in its distribution, but imparted to some tissues more than to others, more histogenic in origin rather than humoral; but not as yet known to be the exclusive property of any particular cell; not necessarily dependent upon what is called allergy; not possible to produce by active or passive immunization; not necessarily dependent upon the persistence of infection for its maintenance; finally, a state, the mechanism of which is imperfectly understood.

Consistent with increasing knowledge of the rôle that lipoids play in immunity, it seems from available evidence that the biochemistry of the defensive mechanism in syphilis concerns lipoids.

Schmmacher and Bergel studied the chemistry of Spirochæta pallida and conclude that the organism is composed largely of a Gram-negative lipoproteid. The original theory that the Wassermann reaction is the expression of an antigen-antibody reaction in the serologic sense has been given recent support through the investigations of Sachs, Klopstock and Weil and others. These workers obtained positive Wassermann reactions in the serum of rabbits after injecting them with mixtures of alcoholic extracts of heterologous and homologous organs and hog serum. The results of these studies suggest that there is concerned in the mechanism of the Wassermann reaction an antibody for lipoid, whether lipoidal of Spirochæta pallida or of tissue is controversial.

Bergel emphasizes the rôle of lipoid substances as a source of antibody formation in syphilis. He believes an important defensive reaction in syphilis is the lipolytic enzymes of lymphocytes acting on the lipoidal Spirochæta pallida. Bergel modified the course of syphilis in rabbits by preliminary treatment with lecithin, which he attributed to the formation of nonspecific antibodies.

The idea of a life-cycle of Spirochæta pallida is being revived. Spirochæta pallida cannot be demonstrated microscopically in infected lymph nodes. Levaditi grafted such nodes in the scrotum of rabbits and examined grafts of these nodes at intervals. He observed various forms with a great affinity for the silver stain. These he considered to be possibly stages in the life-cycle of Spirochæta pallida.

Warthin describes granular silver-staining forms in frequent association with perfectly preserved spirochetes in the lesions of latent syphilis. He believes that the different stages of the forms observed, represent changes in the spirochetes.

Kolle demonstrated that rabbits injected with bismuth compound and then inoculated with Spirochæta pallida, fail to develop a

chancre although the organism is found in the lymph nodes. When the bismuth depot was cut out, a chancre developed. He concludes that bismuth only suppresses the development of a chancre without preventing infection. Levaditi, Fournier and others, however, have shown that it is possible to eradicate the infection with bismuth and that Kolle's contention must be considered in relation to absorption of bismuth, which concerns the use of a soluble or insoluble preparation.

Levaditi and others have tested *in vivo* the spirocheticidal action of 45 metals. Of these, 8 were found to be active, namely, arsenic, gold, mercury, bismuth, vanadium, ruthenium, platinum and tellurium. Tellurium is actively spirocheticidal. Clinically, however, its use is limited by reason of the fact that it forms methyl-tellurium, which is excreted by the lungs and imparts a strong garlic odor to the breath. Furthermore, it bleaches the hair and pigments the skin. Vanadium is too toxic, and gold too feebly spirocheticidal to be of practical use.

Comparing toxicity and therapeutic effect in syphilitic rabbits, the arsenicals and bismuth preparations are the most superior spirocheticidal drugs.

Levaditi and his coworkers concluded from studies that bismuth exerts a spirocheticidal action after conversion to a proteometallic compound (bismoxyl). They believe the liver is particularly concerned in this conversion; indeed, mixing liver with bismuth before injecting it, greatly enhanced spirocheticidal action. The therapeutic action of bismuth is proportional to the quantity of activated bismuth in the body. This quantity is indicated by the tissue content of bismuth-tissue metallic potential. Preparations which they regard as ideal are the fat soluble ones. Assuming that their findings are applicable to the treatment of syphilis in man, then their studies show that the choice of compound in the bismuth therapy of syphilis is a matter of considerable importance.

BIBLIOGRAPHY

Brown, Wade H.: Syphilis, Immunity, Reinfection, Superinfection, VIII Congres International De Dermatologie et de Syphiligraphie, Copenhagen, August, 1930, p. 140.

Chesney, Alan M.: Acquired Immunity in Syphilis, The Am. Jour. Syphilis, 1930, xiv, 289.

Harrison, L. W.: Review of Experimental Research in Syphilis and Its Possible Application to Man, British Jour. Venereal Dis., 1929, v, 173.

Klauder, Joseph V.: The Humoral Aspects of Immunity in Syphilis, Arch. Dermatol. and Syphilol., 1931, xxiii, 884.

Truffi, Mario: Immunite, Superinfection, Reinfection Dans La Syphilis, Annales of Dermatologie et de Syphiligraphie, 1930, Series 7, No. 7, p. 689.

RECENT DEVELOPMENTS IN THE SEROLOGY OF SYPHILIS*

By JOHN A. KOLMER, M.D.

(From the Research Institute of Cutaneous Medicine of Philadelphia)

During the past five years the most important development in the serology of syphilis has been a further refinement of various flocculation or precipitation tests for the diagnosis of this disease. Since first described by Michaelis in 1907, a large number have been developed in different parts of the world and especially in Europe, the most widely employed in this country being those of Kahn and Kline.

For many years it has been known that flocculation or precipitation, invisible to the naked eye, but detectable by microscopic examination, occurred during the process of complement fixation and for this reason it has been long suspected that the mechanism of the flocculation and Wassermann reactions in syphilis may be essentially similar. Further evidence of this relationship between the two procedures has been recently furnished by Eagle who declares that there is a specifically altered fraction of the serum globulin which is responsible for the precipitation and complement-fixation reactions. Eagle determined that, when syphilitic serum is added to the antigen in the flocculation and complement-fixation tests, a specifically altered fraction of the serum globulin is adsorbed by the antigen which is irreversible and that the globulin acquires the properties of a denatured hydrophilic protein in suspension which precipitates in the presence of small amounts of electrolytes responsible for the Wassermann and various flocculation reactions.

In view of this physicochemical nature of both reactions it is to be expected that in the conduct of flocculation and complement-fixation tests in syphilis it is impossible in the light of our present knowledge to render the factors exactly the same and for this reason it is to be expected that these serological tests will yield a percentage of discrepant results and especially in the case of those sera and cerebrospinal fluids containing small amounts of syphilis antibody.

* Read May 6, 1931.

At the present time the best flocculation and complement-fixation tests in the hands of experienced serologists yield about 95 per cent of reactions in agreement in so far as positive or negative reactions are concerned, the remaining 5 per cent being composed of reactions in which the flocculation reaction is positive and the complement-fixation reaction negative or the reverse. For this reason it may be stated *that the serology of syphilis is best served at present by conducting both tests with all sera.* As recommended by the Second Laboratory Conference on the Serodiagnosis of Syphilis by the Health Organization of the League of Nations, held in Copenhagen in 1928, at least two different serodiagnostic methods should be used on the basis of the proven fact concordance of reaction to two or more tests has greater diagnostic value than has a single reaction.

In this connection it is to be regretted that overemphasis has been placed on the alleged simplicity of the flocculation procedures since in truth they demand a considerable experience and a high degree of technical skill for their conduct to secure reactions of maximum sensitiveness consistent with specificity. Under certain circumstances they must be relied upon alone for the serum diagnosis of syphilis as on board ships and in small private laboratories, but in the majority of laboratories it is readily possible to conduct the Wassermann test as well and, as stated above, this is always advisable since the flocculation procedures cannot be stated to have replaced the Wassermann reaction but to have established themselves during the past five years as valuable control tests to be included whenever and wherever possible for securing the maximum of accuracy in the serology of syphilis for both diagnosis and as a guide in treatment.

This is especially true in the serum diagnosis of latent or concealed syphilis when but small amounts of antibody are present in the serum yielding doubtful or falsely negative flocculation reactions since weakly positive reactions are much more difficult to read and interpret than weakly positive complement-fixation reactions. Furthermore it would appear that the flocculation tests are more likely to yield falsely positive reactions in the hands of ordinary laboratory technicians than complement-fixation tests although in the hands of experts, the incidence of falsely positive reactions has been remarkably low with both procedures.

Among the various complement-fixation tests in use that of Kolmer has gained wider and wider recognition during the past

five years as possessing the maximum of sensitiveness consistent with specificity although no alteration has been made in the technique of his test since described in 1923 except the routine removal of natural antisheep hemolysin from all sera which has materially increased its sensitiveness while maintaining its exceptionally high specificity for syphilis. The Kahn test however, has undergone some technical changes and especially an increase of sensitiveness in a "presumptive test" which however, is too sensitive and too likely to yield falsely positive reactions for routine use. The Meinicke test has also undergone technical improvement to the point where in the opinion of some serologists, it is proving superior to the Kahn test. And among valuable additions to the list of flocculation procedures within recent years have been the tests of Bass and Johns and Hinton.

But the most noteworthy development within recent years in flocculation procedures has been the Kline microscopic test, as it has apparently increased the sensitiveness of flocculation reactions while adaptable to use with very small amounts of serum. In this connection it is proving of some value in the serum diagnosis of primary syphilis employing chancre secretions since the collection of a few drops is sufficient not only for dark-field examination for Spirochæta pallida, but likewise for a microscopic precipitation test and proving more practical than the complement-fixation test with chancre secretions developed some years ago by Klauder and Kolmer, because requiring less secretion for the reaction.

In the hands of experts all of these serum tests are capable of yielding very sensitive reactions possessing a remarkable degree of practical specificity for syphilis. None are biologically specific for the disease as all employ extracts of nonsyphilitic tissues for antigen and especially extracts of beef heart. But despite this lack of biological specificity, all possess an extremely valuable degree of practical specificity for syphilis in the United States where human trypanosomiasis is unknown, frambesia or yaws rare and leprosy uncommon. Indeed their use urgently requires a complete revision of the earlier literature upon the serology of syphilis since none of them are as likely to yield the falsely positive reactions in the numerous nonsyphilitic diseases so commonly assumed by many physicians on the basis of the defective tests of earlier years.

All flocculation and complement-fixation tests are capable of yielding false reactions because of technical errors but the incidence of

these is remarkably low in the hands of expert serologists and probably not as frequent as clinical errors in the diagnosis of syphilis. Under proper technical conditions they possess an extremely high practical specificity for syphilis in this country. All have been developed to the point of possessing a high degree of sensitiveness and capable of establishing the diagnosis of syphilis in the clinically difficult, latent or concealed stages of the disease when clinical diagnosis may be impossible and the presence of the disease unsuspected.

But despite technical improvements of the past several years resulting in an increased sensitiveness of serum tests, all complement-fixation and flocculation procedures are still capable of yielding falsely negative reactions and especially in some cases of extremely latent syphilis with special reference to late congenital or prenatal syphilis of children and syphilis of child-bearing women. There is still room for a further increase of sensitiveness but only as far as preserving specificity since it is a simple matter to make the very regrettable mistake of increasing sensitiveness to the point where falsely positive reactions occur. And the writer is of the opinion that it is better to miss an occasional case of syphilis than to run unnecessary risks of making a false diagnosis, on the basis of the thought and practice that the serum tests for syphilis should possess only the maximum of sensitiveness consistent with specificity. And with this in mind simplicity of technique along with economy of time and materials should be regarded as of secondary importance only with the general result that the serum diagnosis of syphilis and serological tests as guides in treatment are best served at present by using two or more procedures, one of which should be a Wassermann test of proven sensitiveness and reliability.

BISMUTH IN THE TREATMENT OF SYPHILIS*

By CARROLL S. WRIGHT, M.D.

The discovery that bismuth, a drug previously considered comparatively inert, exerts a powerful action against syphilis marks an advance in the therapy of that disease which is exceeded only by the chemotherapeutic research of Ehrlich, which resulted in the discovery of the arsenobenzenes. Although bismuth has been used in the treatment of syphilis only since 1921, attention was first directed to its possible employment by Balzer in 1889. Balzer experimented with dogs, but the severe toxic symptoms produced by bismuth caused him to abandon his studies. In 1916 Sauton and Roberts, following the publication of their work on the bactericidal action of bismuth compounds, reported on the spirocheticidal action of the drug in hen spirillosis. Their anticipated work on the action of bismuth in the treatment of recurrent fever and of syphilis was interrupted by the World War. It remained for Sazerac and Levaditi to prove the value of this drug in the treatment of syphilis. As a result of their studies they concluded that "bismuth exercised an incontestable therapeutic action upon experimental syphilis," and the next step was the application of bismuth therapy to human syphilis. Since that time bismuth has been almost universally accepted as a powerful weapon in the fight against syphilis, and recognized as a drug which, though inferior therapeutically to the arsenobenzenes, is superior to mercury both in therapeutic value and in its lower toxicity.

The past five years have seen the introduction of hundreds of proprietary bismuth preparations, put up in diverse forms—water-soluble, insoluble, fat-soluble, colloidal and metallic preparations. To choose from these the preparation giving the maximum therapeutic effect with the minimum toxicity is indeed a difficult problem. Fortunately, the literature is replete with excellent papers on the pharmacology of bismuth and a brief summary of a few of these studies may prove helpful. Particularly important are the studies

* Read, May 6, 1931.

of Levaditi and his co-workers, Lomholt, Gruhzit and Sultzberger, and more recently Hanzlik and his co-workers. The average content of bismuth in the products available ranges from only 10 mg. per cc. of colloidal bismuth up to 73 to 100 per cent in some of the metallic bismuth compounds. However, Hanzlik concluded that the magnitude of bismuth content is probably the least important criterion which should determine the choice of a product, and that other criteria such as vehicle, tolerance, absorption and excretion, as well as antisyphilitic efficiency, are much more important. Everything else being equal, the adjustment of dosage will compensate variations in bismuth content.

Collectively, the results given in the literature on the absorption of bismuth obtained by chemical analysis of tissues after the injection of different products indicate that all types of bismuth are well absorbed from the site of injection regardless of the type of vehicle; the poorest absorption occurs with the insoluble products, used in oil or aqueous medium, and the greatest with the soluble products used in aqueous medium. After urinary excretion is completed, roughly one-half of the administered bismuth remains in the body to be gradually excreted over long periods of time. Levaditi's most recent studies have been concerned with fat-soluble bismuth which he believes constitutes a link between the soluble bismuth salts and the insoluble derivatives, resembling the former in rapidity of absorption and spirocheticidal activity, and the latter in the formation of a local deposit with slow dissociation of the fat from the bismuth giving a slow and prolonged therapeutic action. Roentgenographic studies indicate that metallic bismuth may be unabsorbed after 150 days and there is a question whether metallic-bismuth preparations should be used because of the danger of cumulative action. From our present knowledge it would seem advisable to employ water-soluble preparations in aqueous or tissue-soluble medium or lipo-soluble preparations, although some of the insoluble salts have been used with excellent therapeutic results and have been well tolerated.

As to the avenue of introduction of bismuth in the therapy of syphilis, the limitations are much more marked than in the case of mercury. Klauder, Oelze and others have shown that very little absorption takes place through the unbroken skin. It has also been shown that little or none of the drug is absorbed through inhalation. Absorption through the gastrointestinal tract is slow and uncertain.

It is only necessary to consider that bismuth has been used for several decades in the therapeusis of various gastrointestinal diseases and more recently has been employed widely in roentgenographic studies of the gastrointestinal tract with but rare instances of toxic effects to realize its slight absorption through the walls of the intestinal tract. Sazerac and Levaditi found that the administration of bismuth orally had no effect upon rabbit syphilis. They furthermore found that bismuth administered intravenously was many times more toxic than when injected into the muscles. The Council on Therapeutics of the American Medical Association has taken a definite stand against the employment of intravenous bismuth therapy. The consensus of opinion at the present time is in favor of administering bismuth in the treatment of syphilis only by the intramuscular route. The technique of its administration is exactly the same as for the intramuscular injection of mercury.

As to the efficacy of bismuth in the therapy of human syphilis, there is no longer any doubt. It causes the disappearance of spirochetes from local lesions; it results in prompt involution of cutaneous manifestations; it is of distinct value in the treatment of visceral syphilis; at the same time administration of the drug is rarely followed by any untoward symptoms. I will very briefly enlarge upon the effects of this drug in the treatment of early and late syphilis.

Employed in primary syphilis, bismuth results in the rapid disappearance of spirochetes from the surface of the chancre, sometimes within twenty-four hours after the first injection, and almost invariably after the second injection. After three injections spirochetes can no longer be found even by gland puncture. Healing of the primary lesion progresses more slowly than when the arsenobenzenes are used, but the action on the involution of the satellite adenitis has been found to be more rapid with bismuth. The influence of bismuth on the secondary manifestations of syphilis is rapid —almost as rapid as the arsenobenzenes. However, in spite of the excellent therapeutic activity of bismuth in early syphilis, it is best not to employ it alone, but rather to use it as an adjunct to the arsenobenzenes, in accordance with a method suggested six years ago by Schamberg. Weekly intramuscular injections of bismuth are employed for the purpose of forming depots of ammunition from which the spirochetes are continually bombarded between the shots of that more telling but brief-acting weapon, neoarsphenamin.

In the treatment of visceral syphilis bismuth is perhaps the drug of choice. Its action in tertiary hepatic syphilis is slower than that of the arsenobenzenes, but this is desirable. The remedy of choice, particularly in the hypertrophic gummous hepatitis, is one which will cause a slow involution and absorption of the lesions, thus giving an opportunity for the establishment of a collateral circulation. Bismuth is apparently not hepatotoxic.

Since the publications of Brooks, Wile, and Kothay and Müller several years ago it has been generally recognized that it is inadvisable to employ a drug in the treatment of cardiovascular syphilis with too rapid a therapeutic effect. Bismuth is an admirable remedy in this type of visceral syphilis for its action is efficacious and yet not too rapid. One of the most extensive studies of cardiovascular syphilis and bismuth therapy is that of Laubry and Bordet, who found that there was amelioration of the functional trouble as quickly as arsenic preparations would give; that there was an arresting action on the progress of the lesions as confirmed by radioscopic examinations and that tolerance to the drug was better than tolerance to the arsenobenzenes. This has been my experience. I have witnessed the gradual disappearance, as evidenced by serial roentgenographic studies, of an aortic aneurysm under the influence of pure bismuth therapy.

The results of the treatment of neurosyphilis with bismuth depend upon the amount of the drug which penetrates to the central nervous system and the nature of the lesion. This drug is filtered out by the choroid plexus for the most part, but some writers have recorded the finding of small amounts of bismuth in the spinal fluid in patients under treatment. Even though the amounts of the drug that penetrate into the central nervous system must be small, the clinical results from its use are sometimes striking. Early syphilitic meningitis may respond quickly and there may be rapid cessation of syphilitic headache; vertigo, meningoradicular pains and the lancinating pains of tabes are also often quickly relieved. In true general paralysis bismuth may result in temporary improvement, but advanced cases show no response.

Bismuth exerts an incontestably favorable action in the treatment of congenital syphilis. At the congenital syphilis clinic at the Graduate Hospital, where from 50 to 60 children are treated weekly, I have employed bismuth extensively since 1923, with excellent therapeutic results both as regards the effect of the drug upon

active manifestations of the disease and the effect on the Wassermann reaction. The reports of Cabaout, in France, of Bronson and Caffey, in this country, and of others testify to the value of bismuth in the therapy of congenital syphilis. In 1930 Perlman and I reported the excellent therapeutic results following the use of bismuth alone in the treatment of interstitial keratitis. Kleefeld, of France, believes bismuth to be superior to any other antisyphilitic remedy in the therapy of interstitial keratitis. There are practically no ill-effects following the administration of this drug to children and infants, and the use of comparatively large doses causes little local pain.

In the treatment of the syphilitic pregnant woman, where there is no kidney disturbance, bismuth is distinctly the remedy of choice as an adjunct to arsenobenzene therapy. When there is evidence of renal irritation bismuth alone may be employed with little or no increase in the degree of irritation.

Fournier and Guenot were the first to study the effect of bismuth upon the Wassermann reaction and they found that whatever the stage of the disease, the reaction was favorably affected, sometimes becoming negative before the completion of the first series of injections. In 1927 I reported the effect of bismuth upon the blood Wassermann reaction of 128 patients who had had previous therapy with an arsenobenzene and mercury. Of these 128 only 26, or approximately 20 per cent, were Wassermann-negative after an average of 18 injections of arsenobenzene, and 6 injections of mercury salicylate. Of the 102 still Wassermann-positive, 52, or 51 per cent, were serologically negative after an average of 15 injections (of 100 mg. each) of bismuth.

As to the complications of bismuth therapy, they are few. Studies of the absorption of the drug have shown that large doses are unnecessary, and when small doses are employed there are relatively few signs of intolerance. A blue line may develop on the gum margin, but this offers no objection to continuance of the drug. Odontalgia and salivation are rare, but may occasionally mark the beginning of a bismuth stomatitis. Albuminuria may occur but it is rare. So rare are complications from bismuth therapy, that in a series of more than 25,000 injections given in the past nine years at the Graduate Hospital there has never been a severe untoward effect.

Ten years have passed since Sazerac and Levaditi and their

co-workers, Fournier and Guenot, laid down the principles of bismuth therapy, and in a recent paper published in this country Levaditi has reiterated these principles. Although he believes that their truth has been fully proven in the past ten years, I feel that some allowance must be made for the fact that Levaditi is trying to make the strongest possible case for bismuth, which is in a way a child of his own rearing. He gives the following ten principles of bismuth therapy, all attesting as to its value as an antisyphilitic remedy and to its innocuousness:

1. Bismuth has a curative and preventive action in primary, secondary and tertiary syphilis, in the visceral manifestations of syphilis, in acute syphilis of the central nervous system and in certain forms of parasyphilis.

2. It causes the rapid disappearance of the treponema from primary and secondary specific lesions, and sometimes after the first injection, more often after the second.

3. It sterilizes the lymphatic glands as demonstrated by experiments upon animals and by the results of gland punctures performed after the commencement of treatment.

4. It causes a favorable modification of the reactions of the blood and spinal fluid. Applied to preserologic period bismuth therapy can keep these reactions negative indefinitely, which is an indication in favor of the disease being cured. If bismuth therapy is commenced when these reactions are already positive it succeeds in rendering them negative. It is certain that the curative action continues after the physician has ceased the administration of bismuth. Once serum reactions are negative they can be kept negative indefinitely.

5. These data prove that bismuth exercises a profound and lasting curative action in syphilis, due to the fact that, in contrast to arsenic, it is not eliminated rapidly.

6. Bismuth acts where arsenic fails—in cases labeled arsenic-resistant, bismuth displays a remarkable efficacy. (Levaditi should also say that the reverse may be true, that arsenic acts where bismuth fails.)

7. In comparison today, one is forced to admit that arsenic therapy has only one advantage over bismuth therapy: this is, that arsenic, being administered intravenously and absorbed quickly, causes a more rapid disappearance of open specific lesions than bismuth injected intramuscularly. But the difference is one of several

hours, or at the most two days, to the advantage of the arsenic. This has a certain importance from the point of view of the prophylaxis of syphilis, prevention being all the more efficient the more rapidly spirochetes disappear. But this advantage of arsenic is counter-balanced by the disadvantages of arsenic therapy, the immediate or delayed complications, the rapidity of elimination of arsenic and the frequency of relapses.

8. Another advantage of bismuth therapy lies in its absolute innocuousness. Apart from some complications, all curable and easily avoided, nothing very troublesome has been laid to the account of bismuth. This gives the physician a tranquillity he never has when employing the arsenobenzenes.

9. In the opinion of all experimenters, bismuth is superior in therapeutic properties to mercury.

10. Finally, bismuth is efficacious not only in acquired syphilis but also in congenital syphilis.

FEVER THERAPY IN THE TREATMENT OF SYPHILIS*

By JAY FRANK SCHAMBERG, M.D.

Despite the signal therapeutic advances that were made in the treatment of syphilis, late parenchymatous involvement of the brain and cord, in other words, paresis and tabes dorsalis, were up to a decade ago barely influenced by therapy. After paresis had once developed the arsenobenzenes had but little effect upon its progress. Dementia paralytica was regarded as a progressive incurable malady which almost inevitably led to death.

The empiric origin of nonspecific therapeutic treatment in this condition was suggested by the old psychiatric observation that mental diseases were at times favorably influenced or cured by an intercurrent febrile disease. The credit of introducing and developing this line of treatment is due to Wagner-Jauregg, of Vienna.

Hippocrates and Galen and Boerhave and Sydenham recognized the fact of the healing influence of febrile diseases on the course of psychoses. Various infectious processes, such as typhoid fever, typhus, malaria, cholera, variola, erysipelas, scarlet fever, measles, pneumonia and chronic pyogenic affections were known to produce a favorable influence.

It seems that Rosenbloom, of Russia, in 1874 and 1875, observed 4 cases of striking improvement of psychoses after intermittent fever, 6 after typhus and 22 after recurrent fever. There were 11 cures and 3 improvements. It is not clear that any of Rosenbloom's cases, however, were paresis.

But it was Wagner-Jauregg's prolonged and systematic observations and experiments, beginning in 1883, which finally established the efficacy of malarial infection. Furthermore, the stimulus for further research came from an article by him in 1887.[1] He regarded favorably intermittent fever, erysipelas and recurrent fever as processes which could be artificially induced.

After having tried with variable results tuberculin, tuberculin with mercury, Bezredka's polyvalent typhoid vaccine, staphylo-

* Read, May 6, 1931.

coccic vaccine, erysipelas inoculation and other methods, Wagner-Jauregg began malarial inoculation in 1917. Since that time many thousands of cases have been treated by this method in almost all parts of the world.

The results in a general way are in accord with those treated by Wagner-Jauregg and Gerstmann. Speaking broadly, one-third or more of the patients go into good remissions and a large percentage of these are enabled later to resume work. About one-third have partial remissions, some of these later passing into complete remissions. Finally, about one-third are not improved.

In a general way inoculated malaria is attended with a mortality of about 8 per cent, and it depends much upon the selection of cases. The duration of the remissions is not yet definitely known. Some patients have been back at their work now for nine or ten years. The best results with therapeutic malaria are obtained in paresis. Generally speaking, malaria is distinctly less valuable in tabes dorsalis than in paresis. Nevertheless, some very good results have been reported.

Lancinating pains and gastric crises are usually made worse during the course of the paroxysms, but a great improvement takes place later. It would appear that improvement or disappearance of gastric crises and lancinating pains occurs in about 50 per cent or more of cases. Ataxia is usually not much influenced, but occasionally the improvement is striking. Bladder and rectal disturbances often yield to the treatment. In our own cases a relapse of gastric crisis occurred in several patients, an observation noted in an undetermined proportion of cases by others.

Speaking broadly, malarial inoculation may be recommended in appropriate cases of tabes.

Malarial inoculation has been fairly extensively used in early syphilis by Kyrle and by Vohwinkel, preceded and followed by courses of the arsenobenzenes. In view of the excellence of results obtained by approved medicamentous measures, this treatment should not be employed as a routine in early cases but should be reserved for special conditions.

I should say that malarial inoculation is indicated in paresis, certain cases of tabes dorsalis, in some cases of irreducible Wassermann reaction and in cases in which persistently pathologic alterations in the spinal fluid have not yielded to other vigorous antisyphilitic measures. We secured a brilliant and complete cure in an

early arthropathy of the metatarsal bones complicating a resistant cerebrospinal lues.

What are the contraindications to malarial inoculation? The presence of a large aneurysm or marked degeneration of the myocardium would constitute absolute contraindications. Aortitis need not deter one from this treatment. Great obesity would constitute a relative contraindication. A complicating tuberculosis should demand careful consideration before malarial treatment is instituted. Advanced age (over sixty-five years) usually contraindicates malaria and the likelihood of success is lessened in old people. "Galloping paresis" does not do well under this treatment, and likewise far-advanced cases and senile cases and juvenile paresis. Severe nephritis would also cause one to veto malarial treatment.

The serologic improvement in the blood and spinal fluid is evident to the fullest extent, as a rule, only after six or nine months, although pleocytosis and globulin in the latter are largely reduced. There is usually a parallelism between clinical remission and a regression or arrest in the anatomic cerebral processes, as has been determined on persons dying of intercurrent disease. Remarkable changes are observed to have taken place. Furthermore, spirochetes can rarely be found in the brains of paretics after malarial treatment, although they can usually be demonstrated in two-thirds of the untreated cases.

Sodoku, or *rat-bite fever*, was introduced by Solomon and his associates[2] as a method of febrile treatment of paresis. As a therapeutic method it has, according to Solomon, theoretical possibilities equal to malaria. It is too early to evaluate this treatment, but it is doubtful whether it will come into general use.

Plaut and Steiner,[3] in 1919, tried the effect of *recurrent fever* in paresis, influenced to some extent by the biologic parallelisms between the two causative parasites. In general, the theoretic expectations of Plaut and Steiner, particularly as regards the immunity relationships, have not been fulfilled. Most authors agree that the results are far inferior to those achieved by malaria.

Kunde, Hall and Gerty[4] published the results of the use of *intravenous typhoid vaccine* in paresis. Twenty-one out of 49 cases attained a good state of remission. O'Leary, in 1928, treated 26 cases of neurosyphilis by this method. The treatments were less debilitating than after malaria, but the remissions were slower in appearing.

In 1929 Kemp and Stokes[5] treated 65 cases of syphilis with mixed typhoid vaccine. Of this number 2 were paretics and they were not materially benefited by 6 to 16 fever treatments. A group of approximately 46 per cent of apparently irreducible blood Wassermann reactions in various stages of syphilis were reversed by fever therapy alone or combined with arsphenamin treatment.

Schelm[6] used typhoid vaccine in 20 cases of paresis. Six showed definite improvement, 4 moderate improvement and the remainder were uninfluenced. In 50 per cent of the cases the blood Wassermann became negative and in 15 per cent the spinal fluid Wassermann.

The therapeutic mechanism of fever in neurosyphilis is not known. Many theories have been advanced. Gerstmann remarks: "One thing is sure. It is not the fever which is the important curative agency. The temperature elevation constitutes merely one, albeit an important, factor in the vital reaction." In our Research Institute we[7] were not able to protect rabbits against subsequently inoculated syphilis by the intratesticular injection of malarial blood, but we were uniformly able to do so by giving them 14 very hot baths, raising their temperature an average of 4° F. We were able to demonstrate that spirochetic emulsions heated in physiologic salt solution on a water-bath to 40° or 41° C., although showing actively motile spirochetes, either failed to induce syphilis or did so only after a greatly prolonged incubation period. Schamberg and Tseng[8] were able to demonstrate that patients with secondary or tertiary syphilis showed an appreciably favorable effect upon the eruption as a result of extremely hot baths. Mehrtens and Pouppirt[9] gave 900 extremely hot baths to 70 patients during a year and a half and noted favorable results in tabes and paresis.

These results persuade me to believe that the temperature directly or indirectly is the dominant or perhaps the sole factor in the fever treatment of syphilis.

And now a new and most important method of treatment has been developed. Neymann and Osborne[10] have published this year results in the treatment of paresis with diathermy fever. Twenty-five cases were treated, of whom 66 per cent went into clinical remissions. Eight per cent were *markedly* improved. Sixteen patients, or 64 per cent, made an absolute social adjustment and were able to maintain themselves outside the Psychopathic Hospital without supervision.

In the discussion on the paper Koenig, of the Elgin State Hospital, stated that he had treated 50 patients by this method. Of the 48 per cent of improved cases 33 per cent had returned to their former occupations. The incidence of improvement seems to be somewhat higher than after malaria. A temperature of 105° F. or more can be induced and can be readily controlled. There is almost no danger to life from this method of treatment. One must carefully guard against burns by proper technique and careful surveillance. We are employing this mode of treatment and our early results are encouraging. There is relatively little prostration; an hour after the treatment some of the patients saunter about the wards. It will require a considerable period of time to evaluate this method of treatment and to effect comparisons between it and malarial inoculation.

When it is recalled that the making of a diagnosis of paresis was formerly tantamount to signing a death certificate, what great advances have been made!

BIBLIOGRAPHY

1. Wagner-Jauregg: Ueber die Einwirkung fieberhafter Erkrankungen auf Psychosen, Jahrb. f. Psychiat. u. Neurol., 1887, vol. vii.

2. Solomon, Berk, Theiler and Clay: The Use of Sodoku in the Treatment of General Paralysis, Arch. Int. Med., 1926, xxxviii, 391.

3. Plaut and Steiner: Recurrent Infection in Pareties, Ztschr. f. d. ges. Neurol. u. Psychiat., 1919, No. 13.

4. Kunde, Hall and Gerty: General Paralysis: The Effect of Nonspecific Protein Therapy on the Blood and Spinal Fluid, Jour. Am. Med. Assn., 1927, lxxxix, 1304.

5. Kemp and Stokes: Fever Induced by Bacterial Protein in the Treatment of Syphilis, Jour. Am. Med. Assn., 1929, xcii, 1737.

6. Schelm: Typhoid Vaccine in the Treatment of General Paralysis, United States Vet. Bureau Med. Bull., July, 1930, vol. vi, No. 7.

7. Schamberg and Rule: Studies of the Therapeutic Effect of Fever in Experimental Rabbit Syphilis, Arch. Dermat. and Syph., 1926, xiv, 243.

8. Schamberg and Tseng: Experiments on the Therapeutic Value of Hot Baths with Special Reference to the Treatment of Syphilis, Am. Jour. Syph., 1927, xi, 337.

9. Mehrtens and Pouppirt: Arch. Neurol. and Psychiat., 1929, xxii, 700.

10. Neymann and Osborne: The Treatment of Dementia Paralytica with Hyperpyrexia Produced by Diathermy, Jour. Am. Med. Assn., 1931, xcvi, 7.

THE NEWER ARSENICALS IN THE TREATMENT OF SYPHILIS*

Therapeutic Use and Control of Reactions

By ARTHUR G. SCHOCH, M.D.

The discussion of newer arsenicals in the treatment of syphilis will be limited to three drugs, namely, bismarsen (bismuth arsphenamine sulphonate), acetarsone (otherwise known as stovarsol or spirocid), and tryparsamide.

Bismarsen was synthesized in 1925 by Raiziss, and since then it has had clinical appraisal by several investigators. Bismarsen is given intramuscularly in doses of 0.2 gm. in four- to seven-day intervals. Two injections a week are preferable in early syphilis. Its action lies about midway between arsphenamine on one hand and bismuth on the other, and is in keeping with the modern trend of combined arsphenamine and heavy metal therapy.

Bismarsen is slower than arsphenamine or neoarsphenamine in effecting a disappearance of cutaneous and osseous lesions of syphilis, but its effects are lasting and are accompanied by a low percentage of infectious cutaneous relapse.

Papers by Stokes and Chambers, by O'Leary and Brunsting, by Kolmer and by Shivers, up to a year ago, all report favorably on the use of bismarsen in various phases of syphilis.

Very recently Stokes, Miller and Beerman have reported on five years' experience with bismarsen in all stages of syphilis. They find it especially adaptable to the treatment of early syphilis and point out its particular virtues in the treatment of prenatal syphilis and cardiovascular syphilis. They found a very low incidence of treatment reactions both local and general The general reactions resulting from the use of bismarsen in the form of dermatitis, jaundice, hemorrhagic accidents can be summed up by saying that these reactions occur much less frequently than with neoarsphena-

* Read May 6, 1931.

mine or arsphenamine, and when they do occur the extent and severity of the reactions is less than with the other arsphenamines.

Bismarsen, then, lends itself favorably to treatment of all forms of syphilis, probably equal to combined therapy with neoarsphenamine and bismuth in early syphilis, slightly inferior to combined therapy in effecting the healing of cutaneous and bone syphilis and interstitial keratitis, and is often the drug of choice in the treatment of late syphilis in older subjects where treatment reactions would prove more dangerous, yet where the patient should receive active antiluetic therapy.

Tryparsamide has been used in the treatment of neurosyphilis for the last twelve years. Tryparsamide was first given in courses consisting of 8 to 12 injections followed by rest intervals or in alternation with such drugs as mercury or bismuth.

Lorenz recently reported on 317 patients with neurosyphilis treated with tryparsamide and mercury salicylate with clinical recovery in 50 per cent. Of these 152 patients, 87 per cent have remained well for five to six years. Comparing this group of well patients with a similar group of patients that did not recover, Lorenz drew the following conclusions: the patients that responded to tryparsamide therapy and remained well were on the average seven years younger, they had had the disease a shorter time, they presented greater symptomatic abnormality in the form of expansion, depression, mania, etc., they showed a greater gain in weight under tryparsamide treatment, neurological signs of degenerative changes in the spinal cord were much less in evidence, and the spinal fluids of these patients showed marked variations from normal with a more rapid return to normal under treatment.

Lorenz warns against doses of tryparsamide of less than 1 gm. The dose usually given at each injection was 3 gm. The minimum number of injections was 50 at weekly intervals.

Taking the figures of Stokes and Wilhelm, Moore, Robinson and Keidel, Lorenz, Wile and Wieder, and Moore, Robinson and Lyman on serological results obtained in neurosyphilis, and, averaging up their percentages, the following results were obtained: blood Wassermann reactions were reversed in 41 per cent of the cases treated and reduced in 12 per cent. Spinal fluid Wassermann reactions were made negative in 27 per cent and reduced in 62 per cent. The spinal fluid cell counts were reduced to normal in 70 per cent and improved in 25 per cent; the globulin became normal in

32 per cent and was reduced in 24 per cent; the colloidal gold curves in the spinal fluids became normal in 35 per cent and were reduced in 42 per cent.

The past five years' experience with tryparsamide in the treatment of neurosyphilis has shown two outstanding facts to be true: (1) Tryparsamide should be given not in courses with rest intervals, but continuously week after week for at least one year before it is given up. (2) Tryparsamide therapy should be preceded by intensive antiluetic therapy in the form of an arsphenamine combined with either bismuth or mercury where possible.

Acetarsone, otherwise known as stovarsol or spirocid, is the only arsenical which has any therapeutic effect on syphilis when given by mouth. This property alone makes it very adaptable to the treatment of certain types of patients, especially infants and children. Recently Raiziss in this country synthesized acetarsone in a very pure form. This product is much less toxic for experimental animals than the most optimistic reports of earlier continental investigators

Clinically acetarsone has been used by continental clinicians in the treatment of syphilis, especially in infants and children, with great success. Oppenheim, of Vienna, is conservative in dosage prescribed. He gives from 10 to 200 mg. daily in a single dose to infants, depending on the age. The drug is given in milk or weak tea on an empty stomach, for three to five days, followed by a rest period of the same length. He feels that low dosage and broken broken intervals are responsible for his low incidence of toxic reactions. Lesser, of Berlin, uses higher doses and gives the drug daily until the blood Wassermann becomes negative. This constitutes the first "Kur." After a short rest period a second kur is given equal in total dosage to the first. He has obtained excellent therapeutic results with this method. Both Lesser and Oppenheim advocate combining bismuth or mercury with acetarsone if interstitial keratitis is present.

Acetarsone is not restricted to the treatment of syphilis in infants and children. It can be used in adults. Now that the drug is available in a highly purified form, it should soon become recognized as a valuable drug in the treatment of syphilis.

The Prevention and Control of Treatment Reactions. The Jarish-Herxheimer reaction, or accentuation of the diseased process following the first injection of an arsphenamine, and the therapeutic paradox, *i. e.*, the accentuation of symptoms and signs

as a result of fibrosis from too rapid healing, sometimes seen in cardiovascular and hepatic syphilis, are too often forgotten. They may both be avoided, as a rule, if the patient is first prepared with more slowly-acting drugs such as bismuth or mercury and iodides before starting treatment with one of the arsphenamines.

To inject slowly when giving neoarsphenamine intravenously is the single best refinement in technique used to minimize treatment reactions, notably gastrointestinal reactions, and nitritoid crises. Using freshly prepared neoarsphenamine which has *not* been exodized by bubbling throughout the mixing process is also an essential, but treatment reactions occur more frequently when the solution is injected rapidly than when it is injected very slowly. Heyman and Hirshfeld have described a syndrome designated as "speed shock," resulting from the rapid intravenous injection of fluids. This syndrome includes a fall in blood-pressure and respiratory irregularity and is dependent solely on the rapidity of the injection.

After giving intramuscular injections of either soluble or insoluble preparations, such as bismuth, bismarsen or mercury, vigorous deep massage of the buttock immediately after the injection, followed by hot sitz baths to be taken the same night by the patient at home, does a great deal to minimize the discomfort and to preserve the injection site for the large number of injections that are usually required.

In controlling some of the less serious but very disagreeable reactions resulting from intravenous injection of the arsphenamines, several methods have been found of value. Giving 50 mg. of ephedrin a day for two days preceding the injection often entirely eliminates gastrointestinal upsets. Large doses of atropin sulphate hypodermically, $\frac{1}{75}$ to $\frac{1}{100}$ grain, just ten or fifteen minutes before the injection, giving the arsphenamine in divided doses (Bezredka technique), or the use of 10 cc. of calcium gluconate intravenously or intramuscularly just prior to the treatment often prevents the gastrointestinal reactions and nitritoid crises. A new drug recently brought out by Schamberg for the treatment of arsphenamine dermatitis is calcium thiosulphate. The highly rational combination of calcium with the detoxifying action of the thiosulphate radical may prove to be an excellent prophylactic for treatment reactions from arsphenamine other than arsphenamine dermatitis.

Arsphenamine dermatitis is probably the most frequent of the more serious complications resulting from the injection of one of the

arsphenamines. Experimental evidence in support of the hypothesis that arsphenamine dermatitis results from an acquired sensitization to a radical common to all of the arsphenamine preparations is accumulating.

The patch skin-test done with neoarsphenamine solution has recently been shown to detect manifest hypersensitiveness to the arsphenamines. If a patient gives a history of having had an arsphenamine dermatitis, or if he develops an eruption while under treatment with an arsphenamine, a patch-test should be done before continuing arsphenamine treatment. If the patch-test is positive, no further treatment with an arsphenamine should be attempted. If the patch-test is negative, further treatment with an arsphenamine may be carried out.

THE NEWER CONCEPTIONS OF THE DIAGNOSIS AND TREATMENT OF EARLY SYPHILIS*

By VAUGHN C. GARNER, M.D.

The trend of modern medicine is to concentrate every effort on early diagnosis and immediate treatment, and in no disease is the issue so vitally paramount as in the field of syphilis. Studies indicate that there is a 35 per cent increased possibility of permanent arrest of a syphilitic infection when it is recognized in the primary stage prior to the development of a positive Wassermann reaction. While many patients first report too late to achieve this ideal, the following diagnostic hints are suggested to assist in the recognition of primary syphilis:

1. Time has shown the wisdom of abandoning clinical impressions in the interpretation of the suspected lesion. The chancre has no invariable morphological characteristics. While time-honored clinical criteria as indolence, induration, and associated adenopathy are the suspicion arousers of syphilis, particularly at an extragenital site, they are never in themselves conclusive and the burden of proof under all conditions rests on laboratory procedures.

2. The dark-field examination for the Spirochæta pallida has been shown to be the only way to identify early syphilis in the seronegative phase. Some experience is necessary to distinguish the pallida from saphrophytic spirochetes and dark fields from mouth lesions are notoriously unreliable except under expert interpretation.

3. In the presence of a negative dark field, the syphilologist advises the examination of the secretion secured by aspiration from the base of the lesion or enlarged satellite lymph glands. It is advisable to reëxamine the serum from the lesion itself after twenty-four hours intermittent application of salt solution dressings. A base line blood Wassermann test should be made at the first visit.

4. In cases negative to dark field by direct examination as well as aspiration, the blood Wassermann should be repeated at weekly

* Read May 6, 1931.

intervals for two months and then monthly for an additional two months. It is to be expected that about 14 per cent of those previously negative to dark field will yield positive blood tests.

5. The diagnosis of chancroid should be made only in retrospect after a four months' examination for syphilis has yielded negative results. Local treatment on the suspected lesion may be instituted after a twenty-four hours futile attempt to prove the case by dark-field examination.

6. Banal genital lesions as scabies and herpes in exposed persons may be the portals of entry for the spirochete and such cases should not be dismissed from observation without final reëxamination for syphilis.

7. Gonorrhea may mask an early syphilis with an insignificant, a meatal, or an intra-urethral primary, hence every gonorrheal infection should have the benefit of a Wassermann follow-up. Twenty per cent of patients with late syphilis when questioned concerning antecedent venereal infection give simply a history of previous gonorrhea.

The diagnosis of early syphilis then requires more laboratory insight than clinical acumen. It demands a deeper understanding of the problem by physician as well as the laity if the optimum conditions for dark field are to be realized, namely, the examination of early, clean, untreated lesions. Since 65 per cent of all genital lesions are due to syphilis, every effort should be made to conclusively establish the presence or absence of this infection before a diagnosis of a relatively banal condition is accepted. Laboratory means of diagnosis offer the only trustworthy approach to the entire issue.

When one turns to the problem of the treatment of early syphilis, dogmatism must give place to conservatism, for differences of opinion as to drugs, methods and aims make it impossible to define a standard, universally acceptable treatment program. From the great variety of treatment systems that have been proposed, however, certain principles have recently become more clearly defined and are generally accepted as basic to the scientific, intelligent management of the disease.

1. Most authorities believe an arsphenamine to be an essential part of the treatment in early syphilis. Jeanselme attributes a part of the increased incidence of syphilis in France to the overenthusiasm for bismuth and the relative abandonment of the arsphena-

mines. While most syphilologists concede the therapeutic superiority of original arsphenamine, the technical difficulties associated with its preparation and use have enhanced the practicability of neoarsphenamine which has become the drug of choice except in a few of the larger clinics.

2. The early intensive massing of an arsphenamine as advocated by Pollitzer has been modified to 3 injections of an arsphenamine during the first week or ten days of treatment. The studies of Stokes and Becker showed a 10 per cent lessened incidence of relapse when treatment was pushed during this period. Most authorities at present discredit the advisability of awaiting the appearance of secondaries in order to establish an active immunity and insist upon the treatment of syphilis the moment the diagnosis is accurately established.

3. European practice has long indicated the tendency toward more conservative dosage. Schamberg has been the recent American spokesman for this modification. Large doses definitely endanger tolerance, burden the eliminative organs, and increase complications. Massive doses cannot crush a syphilitic infection; it must be gradually worn out through a long siege of less intensive therapy. It is inadvisable to exceed 0.4 gm. of original arsphenamine, or 0.6 gm. of neoarsphenamine. Women, particularly, and many men do well on the repeated use of 0.45 gm. of neoarsphenamine.

4. Many authorities believe that neoarsphenamine is most effectively given at a three-to five-day interval and private care usually permits such a treatment schedule. In clinic practice a weekly treatment interval is frequently more practical.

5. The minimum amount of arsphenamine necessary for the management of early syphilis is from 30 to 40 injections providing clinical and serological response is satisfactory. The studies of Moore and Kemp and the recent work of Stokes, Besancon, and Schoch, and that of Stokes, Cole, Moore, O'Leary, Parran and Wile show conclusively that relapse is proportional to the number of arsphenamine injections given. In patients with early syphilis receiving from 1 to 8 injections of arsphenamine, relapse occurred in from 75 to 90 per cent, while it diminished to 0 per cent in a group of seronegative dark-field positive cases receiving from 21 to 40 injections of arsphenamine.

6. The present trend is toward the simultaneous administration of an arsphenamine and a heavy metal, usually bismuth. Colonel

Harrison of the British Ministry of Health believes that this concurrent use produces synergistic enhancement of the spirillicidal and resistance stimulating properties of the two drugs and definitely protects against neurorecurrence. While overlap with a heavy metal rather than coincidental administration is permissible with original arsphenamine, many authorities no longer consider it adequate to give a heavy metal merely in alternating courses with neoarsphenamine.

7. No complete rest intervals from treatment are allowable the first twelve to eighteen months of the infection. The arsphenamine phase of the treatment is given in courses of 12 to 15 injections with periods of four weeks' rest between courses during which time bismuth is still continued.

It is generally believed to be advisable to treat every case of early syphilis to an empiric standard irrespective of serological findings. While periodic blood Wassermann reactions are essential as an index of therapeutic response, their change from positive to negative should never be an indication for decreased intensity of treatment or lessened watchfulness for subsequent relapse.

The conception of the short abortive course for cure is obsolete and once a patient is committed to the diagnosis of early syphilis, treatment must be continued uninterruptedly to the maximum determined by large experience and judgment. The report of Chargin based on a group of patients on irregular and desultory treatment is at present unsupported by studies of a similar nature and is not a convincing argument against the prolonged intensive method of treatment generally advised.

8. A spinal fluid examination after six months of treatment is essential to evaluation of prognosis and duration of therapy. In general, in the presence of a negative spinal fluid, treatment should be determined for at least a year from the time of blood Wassermann reversal to negative providing such serological findings are permanent. Abnormal spinal fluids demand a therapeutic approach not pertinent to this paper.

The adequate treatment of early syphilis then demands the long-continued, uninterrupted combination of an arsphenamine and a heavy metal given in conservative dosage and administered simultaneously or at least with overlap. Anything short of this intensive approach paves the way for relapse and the tragic late effects of uncontrolled visceral involvement.

THE PREVENTION AND TREATMENT OF PRENATAL SYPHILIS*

By DONALD M. PILLSBURY, M.D.

A DISCUSSION of prenatally acquired syphilis introduces a more hopeful note than can be found in a review of any other phase of the disease. Prenatal syphilis can, in the opinion of many European and American observers, be eradicated.

I shall consider the diagnosis of prenatal lues but briefly; the past decade has seen little added to the long-known diagnostic landmarks. The more adequate Kolmer, Wassermann and Kahn precipitation reactions have given us more sensitive techniques along with a lower percentage of false positives in the pregnant woman. increasing refinement of roentgenologic technique and greater surety of interpretation in the light of syphilis have been the means of illuminating obscurities in the important groups of bone lesions.

The old concept of an almost uniformly positive blood Wassermann reaction in infected infants during the first two months of life must be abandoned. A single negative Wassermann obtained shortly after birth is apparently without value in excluding syphilis. Boas and Marcus have shown conclusively that the development of a positive blood reaction may be delayed until the sixth month, or even longer. The cord Wassermann is even more uncertain and inadequate. Wells reports 6 infants with negative cord Wassermanns in which congenital lues developed within the first month.

Consideration of the early lesions of prenatal lues will be limited to mere mention of the tendency to localize in the circumoral region, the palms and soles, and the anogenital region; and the frequency of bullous lesions, and the importance of the enlarged spleen in the young infant. In any suspected case blood examination need not be postponed because of the bugaboo of venepuncture in the infant. The adaptability of modern serologic technique in the utilization of as little as 0.05 cc. of serum for the Kline test makes

* Read, May 6, 1931.

the obtaining of sufficient blood a simple procedure; a stab wound is usually quite adequate.

The outstanding late lesions may be catalogued briefly: (1) Interstitial keratitis, the ranking sign of prenatal lues, encountered, according to all recent tabulations, in at least one-half of all late untreated cases; (2) the dental changes, particularly Hutchinsonian incisors and mulberry molars; (3) changes in the bones, especially around the elbow and knee and in the tibia, of the nasal septum and frontal bones. These are the bulwarks of the physical diagnosis of late prenatal lues. The complete Hutchinsonian triad, commonly encountered in the classroom, seems rare in practice. The validity of eight-nerve involvement as a pathognomonic sign has been challenged.

The literature contains an increasing number of nondescript signs enthusiastically advanced as pathognomonic of prenatal syphilis. Almost without exception it would seem that these signs are indicative of maldevelopment in which syphilis may or may not be a factor. An example in point is the complete collapse as a diagnostic point of the Carabelli tubercle. A conscious effort to keep syphilis in the diagnostic foreground in the general examination of every maldeveloped child relieves these debatable signs of their sole function as suspicion arousers.

What must be the focus of attack in the promised eradication of congenital syphilis? Recognition and treatment of the disease in the child most certainly will not attain this end, and attention at once shifts to the source of the child's infection, the syphilitic mother. First, then, we must advocate an attempt at control of conception in the syphilitic female, a measure which Hoffmann has emphasized strongly.

The second and most feasible point of attack is early diagnosis and adequate treatment of the syphilitic pregnant woman. Hoffmann states flatly that adequate early treatment will assure a nonsyphilitic child. At the Royal Free Hospital of London there have been no stillbirths due to lues or syphilitic infants delivered of adequately treated mothers in the past five years. Contrast this experience with the figures reported from England and Wales in 1923, when, of 178,000 fetal deaths, 35,000, or slightly over 20 per cent, were due to syphilis. The outstanding series of Boas and Grammeltoft demonstrate the effect of treatment most graphically. Of luetic mothers receiving no treatment, 96.5 per cent of the

infants were syphilitic and only 3.5 per cent healthy. Of the mothers receiving only mercury before pregnancy 90 per cent of the infants were infected. Of the mothers receiving an arsphenamin before pregnancy 80 per cent delivered infected infants. Of the mothers receiving mercury only during pregnancy 72 per cent of the children were infected; of those receiving arsphenamin before and mercury during pregnancy only 27 per cent of the children showed evidence of syphilis; of the mothers receiving arsphenamin during pregnancy but 17 per cent were infected.

Treatment of the pregnant syphilitic woman seems to be tending away from too conservative methods. Vigorous use of the arsenicals in conjunction with bismuth is essential. The figures of Boas and Gammeltoft would indicate that the arsenicals, because of their proven spirocheticidal powers, should be continued to term. It should be kept in mind that treatment at this time is directed primarily toward protection of the fetus; the resistance of the mother to her syphilitic infection is already at high tide by virtue of her pregnancy.

At what period in pregnancy does treatment become less effective as regards protection of the fetus? The best results assuredly will be obtained by treatment from the beginning of pregnancy. Many reports indicate, however, that rapid decline in effectiveness is not noted until after the fifth month of pregnancy. During the last month treatment probably serves little purpose other than protection of the obstetrician from operative infection.

In view of the overwhelming chance for infection of the infant of untreated mothers with early active or latent syphilis, it is a matter for serious consideration as to how the infected infant of a mother receiving inadequate treatment late in pregnancy shall be handled. Should the probable infection of the infant be regarded as cured or latent? The practice in most American clinics is to await the development of active signs in the child before initiating treatment. There are no data available indicating actual passage of antisyphilitic medication to the body of the fetus, and it seems hardly logical to regard the almost certainly infected infant of a mother with early lues as cured. At the Welander homes for syphilitic children in the Scandinavian countries, where the opportunity for long-continuous observation of the syphilitic mother and child is unparalleled, it is the custom to continue treatment of the infant after birth regardless of the presence or absence of luetic signs.

The treatment of prenatal lues has undergone a marked increase in effectiveness with the development of arsenicals suitable for intramuscular injection. Sulpharsphenamin has proven its value unquestionably in the experience of Osborne and Putnam and of Schamberg and Wright. Bismuth arsphenamin sulphonate is not yet completely evaluated, but is showing promise in the treatment of latent prenatal lues at the University Hospital. Osborne and Putnam state that infants with active syphilis at birth or with bullous lesions seldom recover under any treatment. Treatment of the infant with a positive blood and no physical signs may be fairly definitely systematized, subject, of course, to the restrictions of individual reactions to treatment. The system in vogue at the University Hospital consists of weekly or twice weekly injections of bismarsen varying from 10 to 100 mg. A sulpharsphenamin system used by Schamberg and Wright consists of alternate courses of sulpharsphenamin and bismuth or mercury rubs. Bismuth, although far preferable to mercury, is often not well tolerated locally in very young infants, and for this reason mercury inunctions must be substituted. Neoarsphenamin may be substituted for sulpharsphenamin in older children. Its administration by the superior longitudinal sinus in infants is absolutely unjustified and it is doubtful whether the intraperitoneal injection method suggested by Yampolowsky will find wide favor.

In view of the enthusiastic endorsement of stovarsol or spirocide by mouth at the German Dermatological Congress in 1929, some mention must be made of this method. The drug has long been neglected because of alarming toxic properties in the older preparations, but Levaditi, Hoffmann and Kerl consider it of unquestioned value in the treatment of the luetic infant or the protection of the suckling child of a syphilitic mother. American clinical experience with the drug has been extremely meager.

Interstitial keratitis, as the most frequent and damaging lesion of prenatal lues is, in the opinion of most syphilologists, an indication for the almost simultaneous use of every device of modern syphilochemotherapy. It is the outstanding emergency of prenatal lues. Arsphenamin and bismuth should be given concurrently in maximum dosage in conjunction with iodides by mouth. Nonspecific proteins are often valuable. The lesion is capricious and far from satisfactory in its response, but intensive treatment will often prevent involvement of the unaffected eye. Osborne and Putnam report 30

consecutive cases restricted to unilateral involvement by a concurrent sulpharsphenamin-bismuth system.

The frequency of a positive spinal fluid in prenatal lues varies from 20 to 25 per cent in recent tabulations. Tryarsamide has not been completely evaluated. Various European reports speak highly of the usefulness of malarial inoculation when a positive spinal fluid is uncovered, and all observers agree that the infection is well borne in children. The value of routine early spinal fluid examination goes without saying. A full-blown juvenile paresis apparently still presents a hopeless prognosis.

In conclusion, if I can leave one thought of this brief presentation with you, let it be this, that control of conception in the syphilitic female and early diagnosis and adequate treatment of the disease in the pregnant woman far outweigh in importance any other consideration in this phase of syphilis.

SYPHILIS AS A PROBLEM IN PREVENTIVE MEDICINE*

By JOHN H. STOKES, M.D.

Following the summarizing purpose of this symposium, it is my task to attempt to evaluate five years' gain in knowledge of public health, epidemiological and preventive problems in syphilology. Recent surveys by the United States Public Health Service indicate that there are 643,000 cases of syphilis constantly under medical care in this country, and 423,000 new infections annually. The amount of latent, unrecognized syphilis can only be guessed at. Highman estimates that there are not enough physicians in the country to place all cases under medical care. A series of surveys to determine upward or downward trend has led to a considered statement recently made by Dr. Thomas Parran, formerly head of the Division of Venereal Diseases of the United States Public Health Service, and now Health Commissioner of the State of New York, which must come to Americans with a shock. Though syphilis is decreasing rapidly throughout most of Europe, it apparently is not decreasing in this country. It is said to have been reduced in incidence in Belgium, for example, to about one-tenth of its former prevalence. Why not here? In the registration area of the United States since 1920 there have been 500,000 or nearly one-third more cases of syphilis than of diphtheria; three times as much syphilis as smallpox, and five times as much syphilis as typhoid fever. There have actually been 35,000 more cases of syphilis reported than of scarlet fever; 79,000 more than of all forms of tuberculosis; to say nothing of unreported cases.

Syphilis is, then, a critical, perhaps the critical health problem of our time. The difficulty of epidemiological interpretation is conceded and Sequiera has recently reviewed the debatable aspects of the questions involved. But the figures from England, Germany, Belgium and Scandinavia are on the side of expectancy based on the logic of the situation. Infectiousness is controllable by modern

* Read May 6, 1931.

treatment when applied by organizational methods, under the direction and control of the state. Colonel Harrison, the benevolent despot of British venereology, has presented both indirect and direct evidence to indicate a decrease of five-sixths in the incidence of new infections with syphilis in Great Britain since 1918. A colossal survey of the German situation, reported by Jadassohn and others, indicates a reduction in gonorrhea and congenital syphilis between 1919 and 1927 of about one-third; in primary and secondary syphilis, two-thirds; in chancroid, nine-tenths. Such horizontal reductions raise important technical questions and some doubts impossible to consider here. In the Scandinavian countries there is now so little syphilis that specialists are forsaking the field.

Your essayists have indicated the many uncertainties that beset the curability of syphilis in the individual patient. Between the experimental evidence for superinfection; the contestability and rejection, in recent studies, of the overwhelming majority of reported reinfections in man; the effect of such work as Eagle's in increasing the sensitivity of serological tests to the point where "cured" cases appear merely as arrest; and such spirochete-hunting researches as Warthin's which illuminate the remarkable symbiosis of the Spirochæta pallida and man, it is an open question whether treatment does more than induce latency. Fortunately, for the time being at least, suppression of syphilis by sterilization of contacts and carriers has a better outlook. It is the control of syphilis by treatment of the infectious person that has contributed to the declines in incidence above described.

What are the essentials of a program for the extinction of syphilis? Seriatim, they may be outlined as follows:

1. *Dectection of the Disease Before the Blood Serological Tests Become Positive.* Nine-tenths of laboratory research since 1907 has been headed in exactly the opposite direction. As a result, we have today nothing but the technically difficult and inaccessible dark field with which to grasp our "golden opportunity" for the arrest of the disease.

2. *Detection of the carrier by simplified routine tests,* usable in all well-equipped medical offices, on all patients, as are urinalyses and blood counts. The development of the highly efficient Kline slide precipitation test in this direction is most hopeful and important, the positive result to be checked with complement fixation and precipitation tests combined.

3. *Tracing of the Source of Infection.* In this the practitioner is helpless, rather than indifferent, and needs state aid.

4. *Unwavering adherence to the arsphenamines* in the treatment of syphilis during the first five years of its course, and their application without delay and in spite of the blandishments of bismuth, to the chancre and secondaries as soon as diagnosed. The French are apparently paying the price, in part, of a substitution of bismuth for the arsphenamines in early syphilis. The most massive case material yet analyzed, which was presented before the International Congress at Copenhagen last August from American sources, apparently proves beyond further doubt that it is the arsphenamines rather than the heavy metals that control the infectiousness of syphilis.

5. *Prevention of Infectious Relapse.* The investigation just referred to was able to indicate just how much arsphenamine and heavy metal is called for in the reduction of early syphilis to non-infectious latency. The critical point falls between the fifth and the ninth "606" injection, and 20 injections of each drug may be regarded as a public health minimum.

6. *Adherence to System.* Individualization is for late, not for early syphilis. Within the next several years, it is expected that the League of Nations Health Organization studies of syphilis treatment, of which the American investigation now being conducted by five university clinics, the Public Health Service and the Millbank Fund is a quasi-independent part, will propose standard systems, based on world experience, with which every case of early syphilis can be treated by existing means to secure maximum preventive and curative results.

7. *Simplification and Reaction Prevention in Treatment Methods.* The pyramiding of complexities, technical difficulty and painful and dangerous reaction represented by modern treatment methods in unskilled hands is easily the most serious drawback to their effective application today. Toxicity reduction at the expense of therapeutic effect; the rush into popularity of specialists' methods, such as fever, as against tryparsamide therapy; the critically essential spinal puncture, poorly and rarely performed; the pain of improperly given and irritating intramuscular, the penalties of maladministered arsphenamines, have kept practitioners' treatment at a low level of effectiveness. The universalization of effective

treatment must come through simplification of method as well as better technical training of the physician, present and to come.

8. *Lower Costs.* Syphilotherapy shares with medicine at large the serious problems of this field. Their adjustment cannot be discussed here.

9. *Centralized Versus Individual Treatment Control.* The methods of attack which are now producing reductions in incidence of syphilis abroad, are *centralized*. They depend upon the clinic as against the general practitioner. They are the epitome of state medicine as in Great Britain; or *represent socialized insurance* medicine, as through the Krankenkassen in Germany. They have back of them an influential body of popular sentiment, organizations like the Social Hygiene Council, government-subsidized, in Great Britain; the Gesellschaft fur Bekaempfung der Geschlechtskrankheiten in Germany. An order from a director sways a whole movement, determines along modern lines the entire policy of a nation with reference to an infectious disease. In the United States and France, medical practice in this field is still individual. Jeanselme summarized as contributory to the unenviable record of France, the vagary-chasing of the practitioner, trying this drug and that method on an "I-had-a-case" basis; the disposition of the doctor and the patient to unite on insufficient and symptomatic treatment, and the mistaken use of bismuth to replace arsphenamin in early syphilis. Why the almost equally unenviable record of this country? In part, perhaps, because, in spite of 671 clinics for venereal disease, of which 445 are state-aid, 70 per cent of syphilis in the United States is in the care of individual practitioners. Fortunately for the hope of an American solution outside of socialized medicine, we have a body of semispecialists who treat 60 per cent of our venereal disease along lines that can be improved and almost undoubtedly will be. The question today is: "Will they be improved fast enough, and can the sum total of their improvement equal the performance of organization; of clinics supported by the state, by insurance agencies, by private initiative?" The American situation is marked today by the difficulty of popularizing complicated modern methods with general practitioners and the lethargy of a public unaroused and uneducated to the magnitude and gravity of the venereal disease problem. Private corporate initiative is invading the field, amid denunciations from an organized medicine which will not organize itself to meet this situation. A great pussyfooting, under various

names, toys with or evades critical medical issues, leaving disease prevention too often to its fate. The pharmaceutical manufacturers, every man for himself, strive, compete and too often foist. A scattering of voices raises an appeal for an American solution; the practitioner, aided by the state but not submerged by it, in the control of syphilis through the individual treatment of the infected person to noninfectiousness by his individual physician. Can we not preserve a distinguished individualistic tradition in this over-organized world? A farsighted public health officer, the Commissioner of Health for Massachusetts, will tomorrow night deliver the sixth Hermann Biggs Memorial Lecture before the New York Academy of Medicine. His title is almost clairvoyant in its suggestiveness for the situation we have been discussing in the past ten minutes. "Will the Practitioner Practise Preventive Medicine?" In syphilis, let us hope and believe, that he will.

VASOVAGAL SYNCOPE AND THE CAROTID SINUS MECHANISM, WITH COMMENTS ON GOWERS' AND NOTHNAGEL'S SYNDROME*

THE MARY SCOTT NEWBOLD LECTURE
LECTURE XXVII

BY SIR THOMAS LEWIS, M.D., F.R.S.
LONDON, ENGLAND

(From the Department of Clinical Research, University College Hospital Medical School, London, England.)

IN the present lecture I propose to deal with a form of fainting for which the term vasovagal syncope will be found most appropriate. It forms a common and interesting disturbance of a transient kind. It will be described and discussed in relation to a mechanism that has been under close investigation by physiologists in recent years. There is another and distinct condition with which terminology tends to confuse it, namely, what I shall call for convenience Gowers' syndrome; there is a third condition probably comprising a vasomotor disturbance, namely, Nothnagel's syndrome and thought by some to be identical with Gowers' syndrome. The available evidence relating to these last two maladies will be considered sufficiently to allow us to see what there is in it of significance from the standpoint of the vascular system.

Vasovagal Syncope. Thirteen years ago my colleague, Dr. T. F. Cotton, and I[2] became interested in what may be called conveniently common fainting attacks or, more precisely, vasovagal syncope. Such attacks, needless to say, had long been known; but although many essays were written in the eighteen and nineteenth centuries upon syncope, actually it was not until the analysis of disordered action of the heart-beat was accomplished that a real basis for differentiating between different forms of syncope, trivial and grave, was laid. I have previously indicated the belief that that very active phase of work, during which cardiac irregularities were sorted

* Read, October 28, 1931.

out, meant far more to medical progress than the mere analysis of disorderly action of the heart-beat; it brought with it a sharpened and more rigid point of view, clear recognition that much of what had stood for knowledge in medicine was hypothesis based on slender foundations, and a more insistent demand that reasoning from ascertained facts should more widely replace speculation. Syncope may be used as an illustration of this changed outlook. Graphic methods of studying the heart's mechanism soon enabled types of syncope due to disorderly action of the heart to be divided off and, because these became well defined in the process they were divided off with finality. Other and commoner types were for the moment left where they were, but not without its becoming manifest by contrast that their nature was almost unknown. Although the form of syncope of which I shall speak was widely regarded as a thing apart, it had received no analysis. The very simplest facts necessary to its isolation and description, such facts as the change in pulse-rate and in blood pressure, were practically unknown, and precisely for this reason it remained undefined and its clinical associations could not be recognized. It became obvious that if progress were to be made it would be necessary to witness these fainting attacks and to make deliberate observations during them. This was done in a number of subjects. These seem to have been the first precise observations of the kind to be recorded and appear not to have been added to materially by other workers. Since our original report similar observations have been made in my department and with the help of these I am able to speak more fully of the types of patient in which the attacks occur.

We studied the attacks first of all where we had unusual opportunities of studying them, namely, in young soldiers invalided to our war hospital in large numbers for a condition since termed "effort syndrome," a state of ill-health in which the normal response to exercise is exaggerated. The fact that we studied the phenomena in these subjects was a matter of little consequence and should not be regarded as meaning that the attacks are peculiar to such clinical cases. Any such study must of necessity start with a given clinical type, to safeguard as far as possible against confusing attacks of different kinds, and the investigation must first establish by analysis, and thus define, a type of seizure. Time enough when that is done to search more widely to ascertain the full clinical associations.

The purposes of the present lecture are to redescribe the common

type of syncopal attack in such a way that it may be clear as an entity, and so that its recognition may be facilitated or assured, and to put forward certain ideas relative to its pathology. It is not a purpose to dwell upon the clinical associations. Nevertheless, these clinical associations will be touched upon first because this method will help to bring before you at once cases with which you are familiar. Actually this field is still largely unexplored, a fact which provides another motive in bringing the clinical association into the foreground.

Although Cotton and I described our type of syncope in young soldiers in ill-health, we recognized then and have recognized increasingly since, that its incidence is wide. It is, in fact, the commonest form of syncope or fainting. It occurs in both sexes; it affects young adults especially, but by no means exclusively. There are states of the body that can be named as predisposing to it. Thus it is more usual in people who are out of condition or actually in poor health; it is more frequent after recent illness or chronic infection. The presence of fatigue, a period of fasting, confinement to an overheated, overcrowded room are not unusual attendant circumstances. These examples, which will at once recall to you instances within your own experience, belong to the type under general consideration. It is important to say that this statement is based upon the knowledge that the characteristic manifestations presently to be described are actually displayed in these instances. On similar grounds I can state that the same attacks occur in cases of chronic heart disease, including aortic regurgitation. It is of consequence that this should be recognized, for syncopal attacks in such an association give rise to an alarm that is often unnecessary. We are perhaps too apt as medical men to ascribe all symptoms arising during the course of a major disease to that disease, and too disinclined to realize that transient disturbances that not uncommonly yield symptoms in relatively healthy folk also occur in cardiac patients. But the attacks are not confined to people who are unwell or who are under trying conditions of physical environment. They occur also in people in apparently robust health. Frequently the stimulus provoking the attack can be named, it can usually be named when the subject is robust. Nervous agitation and emotional stress, especially if suddenly developed, are commonly provocative; for example, an abrupt order or threat, an unexpected and important encounter, the receipt of unusual and stirring news, the

witnessing of a distressing accident. In healthy people, completely at ease, the provocation may be adequate without arousing feelings either of disgust, fear or dread. In these instances there is usually an element of surprise, as when a subject sees blood flow into a syringe from his own arm, the arm redden in reactive hyperemia or a sphygmograph lever begin to move on his own wrist.

Of syncope occurring, as it is well known it may, in response to pain, I speak a little more diffidently. I have reason to believe that the syncope occurring during the painful phase of coronary thrombosis is usually of the kind discussed, and reason to suspect that syncope during manipulation of disused joints in gallstone and in renal colic is identical; but in the case of these examples sufficient observations have not been made as yet positively to identify the form of syncope; they should be made and that is chiefly why I am instancing them.

Phenomena of the Attack. The description of the attacks may begin with an actual example; others will be found in our original report.[2]

F., aged twenty-one years, was a young soldier invalided from active service. From his schooldays until the time when he was seen he had been unable to play strenuous games owing to breathlessness and early onset of exhaustion. He was invalided for these symptoms, and because he had fainted while on guard duty when attempting to salute an officer of high rank of whom he became aware abruptly. He was an undersized and poorly developed man, presenting no clear signs of disease in heart or other organs.

On December 18, 1916, the patient was sitting and a few cubic centimeters of blood had been withdrawn from a vein in the arm and the needle removed. He was watching the operation and began to feel queer "as though his stomach had turned upside down;" he complained of dizziness, facial pallor was noticed and his head fell forward to his knees. He was placed at once in a long, easy chair and further observed. By this time the pallor was intense and he was restless. The pulse was imperceptible; the heart sounds distant, its rate of beating being 50 per minute; the action was for the most part regular, a single premature beat being noticed. From time to time there were retching movements. The pupils were little if at all dilated. He was limp, mentally confused or actually unconscious for several minutes. A heavy sweat broke out over his forehead, spreading over chest and body. The pallor remained extreme. Respiration was slow and sighing. The pulse was imperceptible for several minutes; as it returned the systolic blood-pressure was registered at 60 mm.; a little later the pressure fell to 55, then to 50; the pulse-rate varying to between 50 and 60. Five minutes after the onset some recovery was noted, the pulse had risen to 64 and the blood-pressure to 80. Nine minutes after

the onset he was able to respond to questions, pulse-rate being 88 and blood-pressure 105. The blood-pressure gradually rose to 110 mm. One-half hour after the man was able to leave the chair and walk unsupported across the room. He was tremulous and tired for thirty-six hours subsequently. His average blood-pressure in ordinary circumstances was 118 and the pulse-rate 80 to 90.

To generalize, the central feature of the attack is the syncope. In some cases the loss of consciousness occurs without warning of any kind and may then result in a heavy fall and injury. More usually the subject is aware that fainting is imminent and sits or lies down deliberately, or slips or falls more slowly and involuntarily to the ground. The warning consists of a feeling of instability or uncertainty, a dimming of vision and a feeling of giddiness. There is never any sense of rotation or of objects swinging around before the eyes; often a very valuable point in differentiating auditory vertigo. The attacks occur rarely, if ever, while the subject is lying down, a point that in the history is often crucial in differentiating it from major attacks of cardiac or epileptic syncope. The standing or sitting position is the rule.* In the syncope here described there may be at an early stage a peculiar feeling in the abdomen, sometimes described as like that experienced in falling rapidly in a lift, or like that experienced in association with nervousness; it is generally accompanied by nausea, sometimes by retching, sometimes by the feeling that the bowels will be moved shortly. In uncomplicated cases I have not known patients to speak of precordial distress or constriction, or of any real alarm. The symptoms that have been discussed all belong to the attack, to describe them as premonitory is merely to say that they occur before consciousness is actually lost; the cardiovascular changes leading up to loss of consciousness have already begun. From the start there is a progressive lowering of blood-pressure and almost always a steep fall of heart-rate, the pulse usually becoming imperceptible and the heart-rate falling to 50 or 40 (sometimes to 20) beats per minute. Accompanying this fall of blood-pressure is pallor, which increases in intensity, ultimately becoming extreme in the face, but being absent or much less evident in dependent parts, such as the hands. The pallor is of the type associated with simple drainage of blood from the skin to dilating visceral vessels.

* There is another and frequent form of giddiness, occasionally leading up to syncope, which is due to a distinct mechanism; it is characteristic of this giddiness that it is usually related to the act of rising to the erect position.

The cerebral manifestations indicate the degree and duration of cerebral anemia, and from the standpoint of the circulation are to be regarded as incidental. When the circulatory changes happen gradually syncope is led up to gradually by feelings of insecurity and dimness of vision, objectively by incoördination, confusion of speech, restlessness, yawning and decreasing response to questions and to physical stimuli; actual loss of consciousness with flaccidity is the rule. If the changes are sufficiently profound the pupils dilate, the conjunctival reflex vanishes; there may be slight clonic movements of the face, upper limb and trunk. The tongue is not bitten, nor is the urine passed. In the severer attack respirations are slow, deep and sighing. Sweating is almost invariable, breaking out on lips and forehead, or over the body generally; usually free, it sometimes drenches the clothing.

Recovery, which is invariable, is generally slower than is the onset; in it the patient is limp, inert and the skin pale and wet. The attack is almost always brief, lasting from two to ten minutes; occasionally it lasts longer; it leaves the patient tremulous, weak and sometimes with severe headache; these after-effects are usually brief, but may last for hours. A large quantity of watery urine is often passed after the attack.

Cause of the Syncope. The slow pulse in the attack is due, as has been shown polygraphically, to a slowing of the whole heart; irregularity, a common feature of vagal slowing, is often displayed. The proof that slowing is vagal is given by atropine, which promptly drives the rate to levels usual under atropine.

The slowing of the heart is insufficient to induce unconsciousness; it is to 50 or 40, exceptionally to 30, beats per minute, rates frequently seen in cases of heart-block, that are without symptoms. The idea that because the vagus is responsible for the slowing, the force of ventricular contractions will be weakened, is open to doubt; appreciable weakening of the beats from this cause has not been demonstrated in mammals; in the dog the heart-rate may be reduced to one-half by vagal stimulation without altering mean blood-pressure. Undoubtedly the main cause of the fall of blood-pressure in these attacks, and the enfeeblement or loss of pulse, is independent of the vagus and lies in the bloodvessels. Atropine, while raising the pulse-rate up to and beyond normal levels during the attack, leaves the blood-pressure below normal and the patient still pale and not fully conscious. Attacks in which the blood-pressure sinks

without lowering of pulse-rate, and the patient verges upon unconsciousness whenever the pressure reaches certain low levels, have been encountered. Thus the cause of syncope is mainly vasomotor and not vagal;* but the vagus adds impressively to the clinical picture by inducing conspicuous slowing of the heart and gastric manifestations.

The combination of vagal and vasomotor effect, while fully justifying the term vasovagal, brings us to consider a discovery that is possibly very relevant to the pathology of the attacks now discussed.

Carotid Sinus Reflex. In recent years a very great deal of interest has been aroused in a mechanism connected with what is now called the *sinus caroticus*. This mechanism has been shown to exercise a powerful controlling influence upon heart-rate, blood-pressure and respiration, and especially in regulating the circulation to the brain. A rise of pressure within the carotid sinus results in a reflex lowering of heart-rate and of systemic pressure; the former is produced through the vagus and the latter through vasomotor nervous channels (Hering[8]) and through an effect on suprarenal secretion (Heymans[9]).

This important discovery has come to us originally, as so many have, through clinical observations. Hering is, in the true sense of the word, the discoverer, in that his careful and prolonged researches first gave proof that a special mechanism exists. Hering, as he himself has told us, was led to this investigation during the period when he was carrying out his important clinical observations upon patients suffering from irregularity of the heart. He was struck by the lightness of pressure upon the carotid sheath that sometimes suffices to produce slowing of the heart-rate, and it seemed to him improbable that the latter could result from stimulation of the vagus. The fact that pressure upon the carotid region of the neck will slow the heart had, of course, been known for many years previously, and such pressure has often been referred to, though incorrectly, as Czermak's experiment.[3] This physiologist published his observation upon pulse slowing in 1866; both he and Waller,[20] whose publication came four years earlier, attributed the

* In our previous account the part played by the vasomotor system in inducing this form of syncope was insufficiently appreciated. The statement therein contained that the attacks may be relieved by atropine is inexact; the slow rate is relieved, but atropine does not promptly abolish pallor or bring blood-pressure back to normal.

effect to vagal stimulation. But the original observation is neither Waller's nor Czermak's; it is Parry's. This Welshman, famous as clinician and experimenter, wrote, in 1799,[15] as follows:

"In patients whose hearts have been beating with undue quickness and force I have often, in a few seconds, retarded their motion many pulsations in a minute, by strong pressure on one of the carotid arteries. An instance of this kind occurred in the case of the late Admiral K. S. His pulse was quick, and already very full and tense. Pressure on one carotid almost instantly reduced the pulse 15 or 20 beats in a minute, without having previously increased either its frequency or strength, and the pulse returned to its former state as soon as the pressure was removed."

Hering showed that the carotid sinus contains the receptor part of the mechanism and that the glossopharyngeal nerve conveys the impulse to the medulla; here the impulses exert two chief influences, they inhibit the heart and they depress vasomotor tone. This remarkable and seemingly purposive grouping of reflex cardiovascular effects also occurs when the depressor nerve is stimulated (see Kisch[12] and Heymans and Bouckaert[10]); the grouping must surely depend upon a close linkage of the efferent channels by the central parts of the nervous mechanism. And, given this linkage, is it not highly probable that the same central mechanism is disturbed in the clinical syndrome of vasovagal syncope, in which strong vagal effects are likewise combined with depressor vasomotor effects?

The suggestion put forward is supported from another source. Pressure on the carotid in man gives rise to gastrointestinal manifestations. According to Waller, a feeling of uneasiness and nausea develop, and increased movements of the alimentary tract are signalled by borborygmi. Danielopolu[4] recorded the movements of the stomach in man and found these at first inhibited and later increased in activity. The associated alimentary symptoms strongly suggest similar occurrences in vasovagal syncope. From the same point of view it would be interesting to know if sweating occurs as part of the carotid sinus reflex.

The evidence derived from respiratory changes is not so clearly in support. Parry first stated, in 1799 (pages 123 and 124) that pressure on the carotid in man causes respiratory distress, or as Waller and Czermak found, longer and deeper respirations (see also Recht[17]). Danielopolu and his collaborators[5] speak of increased rate and depth in man and dogs from the same procedure. But these changes are not quite relevant. The physiological stimulus

of a rise of pressure within the carotid, while decreasing heart-rate and blood-pressure, also decreases the rate and depth of respiration and ultimately leads to apnea (Heymans and Bouckaert[11]). If vasovagal syncope is due to a disturbance of the same central mechanism respiration might be expected to become slow and shallow; but increased depth appears to be the rule. It is probable that the respiratory symptoms in man are not so much reflex as dependent upon loss of blood supply to the head, for they are relatively late manifestations.

If the suggestion proves fruitful and the vasovagal seizures described are indeed set up through the same central mechanism that takes part in carotid sinus reflex, then on clinical grounds it would be legitimate to suggest that this mechanism can be set in motion through many other channels than those as yet recognized of physiology.

Gowers' Syndrome. In the preceding account attacks have been described under the term "vasovagal." There is but one objection to the use of this term: it is that it has been used before by Gowers[6,7] to denote another condition. But the term is so entirely opposite to the form of syncope here discussed and our knowledge of the other condition to which it has previously been applied is so vague, that the earlier usage, which has never become general, can be abandoned with advantage.

Actually there is little real resemblance between the attacks that have here been described and those of which Gowers wrote. His patients were usually women; epilepsy or insanity was sometimes found in the family history; minor epilepsy or migraine was frequently associated. The symptomatology varied and the picture he gave was not very definite. The attack usually lasted one-half hour or more; it would often bring inability to fix attention, *but was accompanied by no impairment of consciousness;* sensations occurred in the epigastrium, but seldom nausea and never vomiting. A sense of coldness in the limbs was frequent, and sometimes an accompanying tingling or a sense of ascending numbness of the limbs, definitely stamped the case as migrainous. Shortness of breath (sometimes intense), precordial discomfort in the form of pain or palpitation, a sense of dread, were among the symptoms. As Gowers says himself, the symptoms were mainly sensory and subjective. The pulse was said to become small, in 1 case almost imperceptible, the face to show pallor, though the latter, judging

from the case reports, was inconstant, rarely conspicuous and sometimes replaced by flushing. Rigors developed in several cases, cramplike spasm in others.

This is obviously a totally different condition from our syncopal attacks and, therefore, dependent upon a quite distinct mechanism; Gowers himself, unlike some later writers on the same subject, clearly recognized the distinction between his cases and those which he terms "simple cardiac faints." It is, indeed, most important they should not be confused; while it is to be said, in justice to Gowers, that he used the word "vagal" as he says, "as a descriptive term, without implying causation," the facts remain that such physiological terms cannot be employed without the corresponding inference being drawn. So it is necessary to state that there is nothing in his description of what he terms "vagal or vasovagal" attacks that justifies the adjective vagal; the evidence actually brought forward consists purely of the subjective feelings of the subject. The pulse-rate was noted in 1 case only and was increased. There is not much in his account to justify the idea of any profound or constant vasomotor disturbance. Actually Gowers thought, though on insufficient evidence, that the vasomotor center was disturbed, causing *constriction* of the vessels. However relevant Gowers' description may be to studies of nervous disorders, it has not helped us to understand cardiovascular syncope or vasomotor disorders, and the continued use of the term vasovagal to indicate these cases drawn from the borderland of epilepsy is in the light of recent work to be deprecated.

Nothnagel's Syndrome. The clinical description given by Gowers was thought by him to resemble an earlier picture painted by Nothnagel,[14] under a term that has likewise caused confusion, namely, "angina pectoris vasomotoria." Their identity appears to have been accepted by more than one writer (Ryle,[18] Collier[1]) on evidence that is suggestive but cannot be regarded as conclusive. I venture to think that further discussion of the matter on the basis of the original reports can hardly fail to prove sterile, since these are scarcely adequate for the final identification of either malady, and that what we require is a closer examination and description of patients of the several kinds while in the attacks. So far as the cardiovascular system is concerned, Nothnagel's description is much more precise than that of Gowers. Nothnagel's 4 patients had no heart disease, 3 of them were men in their thirties

and forties. The attacks were provoked by cold or by bouts of drunkenness. The common signs in the attacks were blanching of the skin, the hands or limbs being involved conspicuously and displaying numbness and coldness. The pulse was narrow and small; its rate was unchanged or dropped. These signs preceded anxiety or actual fear, palpitation and giddiness. The attacks lasted one-quarter to one hour. Consciousness was not lost in them. In 1 of his cases, the only elderly subject, anginal pain appeared. Nothnagel's syndrome is seemingly a rare malady, for no clear records of it appear to have been added since he wrote, unless we include the cases of Powell[16] and Stokes,[19] in both of which, however, the pulse-rate rose (see Lewis[13]). Stokes' case is notable because it is the only one of the series in which actual blood-pressure readings were taken; these were raised.

Nothnagel's evidence of vasoconstriction was chiefly the occurrence of conspicuous pallor and coldness of the extremities. Pallor of the skin, with or without coldness, is in fact inconclusive evidence of vasoconstriction; pallor is a sign of diminished vessels; oncoming coldness, when objective, is a sign of diminished bloodflow. Both these signs can appear when the main vasomotor change is one of vasodilatation in other areas; especially is this true of the facial skin which drains easily, though the limbs may be affected similarly to a certain point. The onset of facial pallor is obviously quite inconclusive evidence of vasoconstriction. The quick onset of *conspicuous* pallor of *hands and feet*, providing these are not held aloft, is a more certain sign of vasoconstriction. Nothnagel stressed the signs of vasoconstriction as the first event, appearing several minutes before the major symptoms. He states this to have been the order of events in 3 of his 4 cases. His was a slender series, but the important view it suggested, namely, that vasoconstriction, by throwing a considerable burden on the undiseased, though not necessarily wholly efficient, heart, can determine secondary symptoms of considerable or severe distress, receives support from a different source. Severe palpitation, great anxiety and apprehension, throbbing in the neck, a sense of oppression in the chest, pain in the chest of anginal type are all symptoms that may be provoked in healthy subjects by the action of adrenalin in man, the symptoms being associated with raised blood-pressure and acceleration of the heart. In paroxysmal hypertension, thought to be due to adrenalin released, a closely corresponding group of symptoms and signs

develop, though intense headache, nausea and vomiting may be added. If we say that Nothnagel's syndrome is consistent with general vasoconstriction and that this would explain the symptoms displayed by his patients, we probably state quite as much as is justified. It seems unreasonable that we should continue to base discussions of vasoconstriction upon what is largely guesswork, while the essential facts are there for the collecting; or that we should continue to be saddled by terms which, by implying more than has been proved, tend to confuse our thoughts. It is difficult to see how a fresh publication of case notes that fail to add essentially to knowledge can make for progress; what is wanted is close study of patients in attacks, with a clear identification of pressure effects and concomitant symptoms. It seems self-evident to state that our understanding of transient derangements of the circulation must depend upon our directly ascertaining whether the blood-pressure rises or falls and what happens to the pulse-rate. Yet these, more often than not, are just the data that are not available, and so knowledge of vasomotor changes in man remains obscure.

REFERENCES

1. Collier: Lancet, 1928, i, 645.
2. Cotton and Lewis: Heart, 1918–1920, vii, 23.
3. Czermak: Jena Ztschr. f. Med. u. Naturw., 1866, ii, 384.
4. Danielopolu: Wien. Arch. f. inn. Med., 1929, xvii, 381.
5. Danielopolu, Mareu, Proca and Manescu: Ztschr. f. d. ges. exper. Med., 1930, lxx, 268.
6. Gowers: Lancet, 1907, i, 1551.
7. Gowers: The Borderland of Epilepsy, London, 1907.
8. Hering: Verhandl. deutsch. ges. f. inn. Med., April, 1923; Die Karotissinus reflexe, etc., Dresden and Leipzig, 1927.
9. Heymans: Arch. internat. d. Pharmacol. e. d. Therap., 1929, xxxv, 269.
10. Heymans and Bouckaert: Compt. rend. Soc. de biol., 1928, xcix, 1871.
11. Heymans and Bouckaert: Jour. Physiol., 1930, vol. lxix, Proc. XIII.
12. Kisch: Ztschr. f. d. ges. exper. Med., 1926, lii, 499.
13. Lewis: Heart, 1931, xv, 305.
14. Nothnagel: Deutsch. Arch. f. klin. Med., 1867, iii, 309.
15. Parry: An Inquiry into the Symptoms and Causes of the Syncope Angiosa, Commonly Called Angina Pectoris, 1799, p. 102.
16. Powell: Allbutt's System of Medicine, 1910, vi, 183 (Case 5).
17. Recht: Klin. Wchnschr., iii, 916.
18. Ryle: Lancet, 1931, i, 737.
19. Stokes: Heart, 1909–1910, i, 297.
20. Waller: Proc. Roy. Soc., 1862, xi, 302.

OSTEOARTHRITIS DEFORMANS CONTRASTED WITH OSTEOARTHRITIS DEFORMANS JUVENILIS*

THE MARY SCOTT NEWBOLD LECTURE
LECTURE XXVIII

BY PROF. F. J. LANG
DIRECTOR OF THE PATHOLOGICAL-ANATOMICAL INSTITUTE OF THE UNIVERSITY OF INNSBRUCK, AUSTRIA

THE comparative study of the adult and juvenile types of osteoarthritis deformans must be based upon a clear interpretation of the anatomic and histologic findings in the frequent and important adult form.

Osteoarthritis deformans is a chronic noninfectious proliferative inflammation that leads to a deformity of the joint structures. The development of marginal exostoses has always been of especial importance for the anatomic as well as the histologic diagnosis of arthritis deformans. These marginal exostoses are characteristic; they are the decisive feature for the diagnosis of this disease (Pommer). The exostoses arise from vascularization and ossification, beginning in the subchondral marrow spaces and invading the overlying noncalcified cartilage. This vascularization and ossification, following the loss of elasticity of the joint cartilage, is the first diagnostic feature of arthritis deformans (Pommer).

These marginal exostoses are not ossified excrescences (Volkmann) nor periosteal or perichondral proliferations or osteophytes (Nichols and Richardson), nor periosteal lippings (Kimura, Ziegler); they are, as Pommer maintains, bone formations that, arising from the subchondral marrow tissue, grow out into the joint cartilage. Of laminated structure, they are united to the spongiosa. Usually they are covered with new cartilage.

The joint cartilage presents various alterations: changes in consistency and splitting of the ground substance. These alterations disturb and damage the elasticity of the cartilage. The margins

* Read, November 4, 1931.

of the fovea and of the head of the femur and, in general, those regions where hyaline cartilage passes over into fibrous cartilage have an especial predisposition for the occurrence of the above-mentioned changes.

A striking sequel to the vascularization and the ossification of the cartilage is the undermining of its deeper portions. The thereby separated portions of cartilage are often found deep in the spongiosa, marking the original boundary between cartilage and bone.

As above mentioned, the periosteum in general does not take any part in the formation of the marginal exostoses. However, in more advanced cases of arthritis deformans periosteal bone proliferations covered by newly formed cartilage can be seen at the insertion of the tendons. The synovial membrane may also be the site of proliferative changes.

Rarely it is observed that, in spite of the changes in the cartilage surface, vascularization and ossification of the basal cartilage layer does not take place. The cause for this is either that the calcification zone of the joint cartilage is very broad or the subchondral bone is very compact. The subchondral bone is thus protected against functional and mechanical damage in spite of the loss of elasticity of the overlying cartilage (Pommer). Hence it is obvious that arthritis deformans is dependent for its development upon the combination of functional and anatomic circumstances.

The layer of cartilage covering large marginal exostoses may be worn away and the underlying bone be exposed and made smooth, giving rise to the polished surface. The increased bone resorption and new formation due to mechanical irritation in those areas of the polished surface, where the bone-marrow lies exposed, render understandable the combination of bone atrophy and bone condensation. In this union of opposite phenomena is found the explanation of the characteristic deformity of the joints in arthritis deformans.

Furthermore, in the more advanced cases of this disease one can find callus and resorptive tissue proliferations, especially in the subchondral bone and along the junction between cartilage and bone. This callus formation arises because of the splitting and cracking of the bone and of the cartilage. These reactive processes make clear the striking picture of porosity and condensation that is so obvious in the macerated specimen.

Microscopic cysts are likewise found in the exposed subchondral

marrow spaces. These cysts are either the result of encapsulation of hemorrhages or of areas of detritus. Thus is disproved the theory of Kimura and Ziegler, that the cause of arthritis deformans lies in regressive changes in the subchondral bone (Pommer). In the far-advanced arthritis deformans so-called cartilage nodules are observed; these are situated in the marrow spaces of the spongy bone beneath the polished surface. According to Pommer, the cartilage nodules can originate in two ways: (1) As callus resulting from microscopic fractures, and (2) as fragments of joint cartilage that are broken off and transported as emboli along the capillaries and lymph vessels growing where they lodge.

With the knowledge of the local origin of the various reactive and progressive changes in arthritis deformans, it can be understood how atrophy, caused by age and inactivity, occurs at the same time that these other changes are taking place. Thus not only sclerotic but also atrophic polished areas are observed.

Considering the above-described findings and their explanation, the distinction between the atrophic and the hypertrophic or between the degenerative and the hyperplastic types of arthritis deformans is not necessary.

According to Pommer and Beitzke, the varieties of arthritis deformans present no distinctive differences; they are but gradations of a process that is uniform as regards its origin. Naturally in the explanation of these differences, individual variations must be considered. These variations have their origin in the anatomic structure, in the functional requirements and in the effacement by use.

A distinction between the chondral and osseous forms of arthritis deformans is also unnecessary. For in the final analysis it is the damage to the cartilage, with its loss of elasticity, that is responsible for the origin of the disease.

In spite of the numerous investigations that clearly prove the inflammatory but noninfectious nature of its origin, arthritis deformans has more recently been called arthrosis, from clinical and roentgenologic points of view.

The designation of arthritis deformans as arthrosis is to be avoided, as the termination "osis" is used in pathology to indicate changes of a degenerative nature. Thus, for example, nephrosis indicates a degenerative change in the kidney, and nephritis an inflammatory process.

It is asserted that constitutional influences have to be considered

in the development of arthritis deformans, and, therefore, the designation arthrosis is preferable to arthritis. This argument, however, is not sound. These considerations are important for every inflammatory process, regardless of what organ or structure is the site of the inflammation. Constitutional influences can, nevertheless, affect the type, the intensity, the extension and the outcome of an inflammatory process.

It is absolutely necessary to keep in mind the histologic distinction between inflammatory and degenerative processes. This distinction is not superfluous and is of value since inflammatory and degenerative processes frequently occur side by side.

Under all conditions the character of arthritis deformans is a chronic noninfectious proliferative inflammation of the joint, and, therefore, the only designation of this process that is justifiable is arthritis.

Regarding the causes of arthritis deformans, it must first of all be admitted that a direct relationship can exist between arthritic changes and certain systemic diseases. But vascular changes, especially arteriosclerosis, are not the main cause for the origin of arthritis deformans. The vessel changes that are sometimes seen are, generally, either independent or secondary to the inflammation or localized circulatory disturbances, such as occur in the advanced cases of arthritis deformans. Likewise there is no proof that, under natural and nonexperimental conditions, the cause of primary arthritis deformans is due to necrotic changes of the cartilage. Furthermore, there is no confirmation of the view that regressive and atrophic changes of the subchondral bone are of importance in the development of this disease.

The only satisfactory explanation for arthritis deformans is found in the functional theory of Beneke and Pommer. This theory presupposes that the joint cartilage, through its elasticity, protects the subchondral bone and its marrow from the irregular, localized effects of impacts, jarrings and pressures arising from normal function. Following damage to the joint cartilage, with subsequent loss of its elasticity, the mechanical effects of function are more or less localized and unmodified, and lead to reactive vascularization and ossification of the cartilage.

The damage to the joint cartilage arises: (1) From unequally distributed or violent strains, for example, of heavy labor, of bent posture or such occurring in tabes dorsalis in which the pain sensi-

bility is lost; (2) through diseases of the joint cartilage itself; (3) through diseases of the joint capsule, the synovial membrane and the subchondral bone; (4) through endogenous metabolic and endocrine influences.

The recognition of the functional and mechanical importance of the joint cartilage as the protective covering of the subchondral bone (Roux) demands that the mechanical-functional damages to the joint cartilage be adjudged the most important factor not only for the pathogenesis but also the etiology of arthritis deformans. An especial confirmation for the functional theory of arthritis deformans is the fact that this disease is frequently found in certain occupations. Sufficient proof for this statement is given in the literature.

R. Beneke has pointed out the possible sequelæ of old traumatic changes. I was able to confirm this idea by my studies of juvenile joints, in which I found evidences of the end-results of more or less extensive traumatic changes. By these studies this theory is substantiated, that arthritis deformans is already initiated in the early years of life, and, therefore, that the disease has to be considered as a consequence of trauma carried over from childhood and adolescence. When the after-effects of trauma for the origin of arthritis deformans are considered in this way it is evident that in the sequelæ of chronic, oft-repeated functional trauma lies the etiology of this disease.

I was able to investigate the juvenile form of arthritis deformans in Legg-Calvé-Perthe's disease of the hip-joint, Köhler's disease of the first and second metatarsal bones and other so-called "aseptic necrosis." In these diseases the characteristic diagnostic features of arthritis deformans were present, namely, changes of the joint cartilage, vascularization and ossification of the cartilage and marginal exostoses.

In the juvenile type of the disease the epiphyseal zone participates in the typical changes found in arthritis deformans of adults. As a sequel to these changes cessation of ossification occurs in the involved portion of the epiphyseal line. In the cases of juvenile arthritis deformans are found striking examples of fissure formations splintering and fragmentation of cartilage, of subchondral bone and of the bone and cartilage along the epiphyseal line. These changes stimulate callus formation, with the production of new cartilage and bone, so characteristic of juvenile arthritis deformans. The

splintering and fissurations are found along the bone cartilage juncture; their lines of direction are in part parallel with and in part at right angles to the lines of strains.

The behavior of the epiphyseal zone and the size, position and structure of the epiphyseal bone determine whether adjustment and distribution of the vibrations of impacts and jarrings are possible, or whether splintering and fragmentation with displacement of the fragments will occur. More or less extensive hemorrhage with subsequent hemosiderin deposits and blood-cyst formation complete the picture of previous trauma.

From my studies it is evident that functional traumas are determining factors for the origin of juvenile arthritis deformans. The continuous effects of this trauma are responsible for the insidious character of juvenile Arthritis deformans.

The following cases of Perthe's disease are illustrative of this point:

The first patient was a normal male, aged twenty-two years, who fell from a ladder, injuring his right thigh. An *x*-ray showed an oblique incomplete fracture beneath the trochanters. After six weeks in a plaster cast the patient was discharged with a negative *x*-ray. Two years later a pronounced flattening of the femur head was visible, which, three and a half years after the injury, was far advanced. At this time, because of pain and limitation of motion, the joint was opened and a mushroom-shaped femur head removed. This case is clarified only by postulating damages to the joint cartilage and, what is especially important, fissure formations in the subchondral bone with a consequent disturbance of the structural equilibrium, changes invisible in the first two *x*-ray films. After six weeks of rest these areas were subjected to continued functional traumas. The final microscopic picture shows tiny pseudoarthroses lined by new cartilage, areas of osteitis fibrosa with islands of splintered and pulverized bone, cysts containing hemorrhages and débris and all the characteristic signs of arthritis deformans.

The second patient was a boy, aged fifteen years, who developed Perthe's disease following a gunshot wound of the soft parts of his right hip-joint, but not involving the bone structure. These *x*-rays were taken immediately, nine and eleven months after the injury.

The bilateral, symmetrical type of this disease of the hip-joint is exceptional in that a developmental malformation of the epiphysis predisposes to functional and actual trauma.

The importance of the trauma for the origin of juvenile arthritis deformans is in no wise lessened because no history of injuries can be elicited. In many comparative studies I was able to demonstrate

histologically in cases giving a negative history of trauma more or less severe traumatic changes with their sequelæ.

When there is a history of trauma without subjective and objective symptoms immediately after the injury one must consider the sequelæ that may be found at a later date. An example of this is the posttraumatic atrophy of the vertebra described by Kümmell. This atrophy may lead to almost complete disappearance and collapse of the body of the vertebræ, but does not become evident until a variable interval of time after the injury.

In estimating the importance of trauma there can be no doubt that the mechanical traumatic damage to a joint due to excessive athletic activity in early life will lead to arthritis deformans.

In conclusion, it is evident that there are no fundamental differences between arthritis deformans of the adult and juvenile arthritis deformans. Both types of the disease originate after damage to the cartilage with subsequent loss of its elasticity. This loss of elasticity removes the protection that the normal cartilage affords to the subchondral bone against functional and mechanical influences.

As a causative agent for arthritis deformans in adults the damage to the cartilage resulting from excessive strains is of prime importance; for the juvenile arthritis deformans the direct and indirect traumatic damages to the cartilage and, especially, to the osteochondral junction is responsible. Decisive for the continued course of both types of arthritis deformans is the action of functional requirements which, acting as a constant irritation, form the basis for the progressive character of the disease.

HOW LONDON AND EDINBURGH INFLUENCED MEDICINE IN PHILADELPHIA IN THE EIGHTEENTH CENTURY*

THE S. WEIR MITCHELL ORATION

BY FRANCIS R. PACKARD, M.D.

AMONG Weir Mitchell's many titles to fame, I think that one of the most outstanding and permanent is that of student and writer on the Colonial period of our history. By many competent critics *Hugh Wynne* has been ranked as the greatest achievement of fiction dealing with that period. The reasons for its success, beyond its charm as a novel, are based on the vividness with which it recalls the life of the time and the accuracy with which it deals with the historic setting. *Hugh Wynne* was the work not only of a brilliant novelist but also of a profound historian, and it bears on every page evidence not only of these qualities on the part of the author, but also of the love he bore for the place of which he wrote and his intimate knowledge of the characteristics of its inhabitants.

One aspect of eighteenth century Philadelphia in which Dr. Mitchell was most profoundly interested was the history of the origins and traditions of the medical institutions of this city, and the lives and characters of the men who established them on the high plane he was proud to think they continued to maintain. It was especially pleasing to him that the source of these institutions could be directly traced to the training received by their founders in London and Edinburgh. Weir Mitchell was of Scotch origin, his paternal grandfather having come to America from Ayrshire sometime between 1786 and 1789.[1] His father, John Kearsley Mitchell, as a little orphaned boy, was sent to some relatives in Scotland, and there received his school education and took some courses at the University of Edinburgh. Before taking up the study of medicine, however, he returned to this country, and his medical

* Read November 18, 1931.

[1] Mitchell, Weir, His Life and Letters, by Anna Robeson Burr, Philadelphia, 1929.

education was entirely received in Philadelphia. Weir Mitchell's own scientific relations with both London and Edinburgh were very close: he was a foreign fellow of the Royal Society; member of the Medical Society of London; honorary member of the Clinical Society of London; and, in 1895, he received the honorary degree of LL.D. from Edinburgh University. He was wont to refer with much pride to the close connection which existed between the early medical institutions of Philadelphia, particularly the Medical School of the University of Pennsylvania and this College of Physicians, and their older prototypes in Edinburgh. In 1887, in an address at the Centennial Celebration of the founding of this College, he said: "Who first suggested the formation of this College is unknown, but, as many of our Fellows were educated in Edinburgh, it is likely enough that the success of its Society, which dated from 1733,[1] may have led them to imitate it here" and, speaking of the Founders of the College, he said: "As to their medical education, the best of them had been educated in Edinburgh, which school is the parent of our University. Genealogically we might speak of our College and of the University as children of Edinburgh and grandchildren of Leyden."

When I was honored with the invitation to deliver the Weir Mitchell Oration, I could think of no subject more conformable to the mind and spirit of the great man whose memory we are honoring than a consideration of the development of scientific medicine in this city and of the English and Scotch influences which played the chief part in it. I shall ask you, therefore, to go back with me to the Philadelphia of the last half of the eighteenth century, when it was the center of medical education and science on this continent, and to glance also at the conditions in London and Edinburgh which led young men to make the then long and difficult voyage across the ocean to avail themselves of the opportunities they afforded.

In 1750 Philadelphia's population was 16,000, which had increased by 1780 to 25,000. We doubt if many cities of its size could show as large a proportion of institutions of learning or a larger proportion of citizens interested in science or culture. The Philadelphia Library had begun its career of usefulness in 1731, the College of Philadelphia (now the University of Pennsylvania) in 1749, and the Pennsylvania Hospital was founded in 1751, and we should

[1] According to J. D. Conrie, "The History of Scottish Medicine to 1860, London, 1927," the Medical Society of Edinburgh came into existence in 1734.

recall that the success of all these institutions was chiefly due to Benjamin Franklin, and that to him we also owe the founding of the American Philosophical Society in 1768, to which most of the Philadelphia doctors of whom I shall speak belonged and in whose affairs many of them took a most active part. Philadelphia can boast of three important "firsts" in the history of medicine in the British Colonies: (1) the first hospital, the Pennsylvania, founded in 1751; (2) the first public medical library, that started in the Pennsylvania Hospital in 1763; (3) the first medical school, established by the College of Philadelphia (now the University of Pennsylvania) in 1765. That these foundations succeeded so eminently in the objects for which they were designed must be attributed chiefly to the wisdom and foresight of their founders and to the training and ability of the physicians who served them, and I hope to show how largely the medical education of these men was due to the eminent teachers then flourishing in London and Edinburgh and how the great traditions of their schools were directly inherited by the earliest medical institutions in Philadelphia and by them transmitted throughout the entire country. This is no idle boast, because though many men from other colonies, such as Samuel Bard, of New York; James Lloyd, of Boston, and Alexander Garden and Alexander Baron, of South Carolina, studied abroad and used their opportunities to forward medical education in their various localities upon their return, the medical institutions of Philadelphia were successfully launched before any others were underway elsewhere. Thus Samuel Bard was the chief factor in founding a medical school in connection with King's College, New York, but it did not come into existence until two years after the medical school in Philadelphia, and it was completely disrupted by the Revolution and only recommenced as the medical school of Columbia College in 1787. The medical school at Harvard was not founded until 1782.

A curious feature in the history of medicine in this country is the variations in the tendencies of the young medical men who go abroad for study as to where they go. There are certain well-defined periods in these migrations. During the Colonial period and down to 1800 the majority went to London and Edinburgh, partly for the obvious reason that they regarded Great Britain as their mother country, but I think more especially because at that time London and Edinburgh were at the zenith of their fame as

medical centers, for reasons to which I shall refer later. When France had settled down after the turmoil of the Revolution and the Napoleonic régime, Paris became the great medical center of the world. Osler has written delightfully of the young Americans who flocked to that city to study in the clinics and laboratories under Louis, Broussais, Dupuytren, Velpeau and other great teachers. It was under Louis that W. W. Gerhard received the training which he turned to such good account later when he studied his cases at the Philadelphia Hospital and discovered the essential difference between typhus and typhoid fever. After the middle of the last century the tide turned and until the Great War the majority of American students went to Germany or Austria for post-graduate work.

Let us begin by considering for a moment the advantages offered our young Colonial students in London. In surgery the great Cheselden, who was especially famous for his skill in operating for cataract and for stone in the bladder (he could perform the lateral operation for stone in fifty-four seconds), died in 1752. His name is embalmed in the verse of Alexander Pope:

> "I'll do what Mead and Cheselden advise
> To keep these limbs and to preserve those eyes."

He had trained many students and his pupils and successors carried on his fame, and the London School of Surgery maintained its reputation, with Percival Pott, Cline and John Hunter, down to the days of Astley Cooper, Abernethy and Brodie. John Hunter was not only the greatest surgeon of this group, but also one of the greatest anatomists of all time. His elder brother, William, had started an anatomical school in London in 1746. John joined him two years later, and from 1748 until William Hunter's death in 1783 and John's ten years later these two men taught anatomy as it had never been taught before. John Hunter seems to have been particularly well disposed toward young Americans. Among those he took into his home as house pupils were Francis Kinloch Huger, the young South Carolinian who subsequently made such a gallant attempt to help Lafayette escape from his prison at Olmutz, and Philip Syng Physick, of Philadelphia. In midwifery, London offered splendid opportunities to the student. William Hunter was not only a remarkable teacher of anatomy, but also of midwifery. His magnificent work on *The Gravid Uterus* was the only medical book

printed at the famous Baskerville press. Another very able teacher of midwifery was George McKenzie, who enjoyed an especial popularity with the American students. Although there were many excellent physicians in London, to some of whom I shall refer in a few minutes, the opportunities for the study of "physic," as it was then called, did not compare with those to be got in Edinburgh.

Now let us turn to Edinburgh and see what advantages it had to offer to the student. It was in no narrow spirit of nationalism that Weir Mitchell spoke with such pride of the filiation of our medical institutions to those of Scotland. It was because he knew that in the last half of the eighteenth century medical education at Edinburgh was on a higher plane than anywhere else in the world. I can only refer to a few of the famous men who were in its faculty during that period. Among them was:

Robert Whytt (1714–1766) who was not only a great physiologist but also a great physician. In his treatise entitled *Observations on Dropsy of the Brain* he gave the first accurate description of tuberculous meningitis, and his treatise on hysteric disorders is ranked as a medical classic. He was succeeded in the chair of practice of medicine by the illustrious William Cullen (1710–1790). Though not distinguished by any particular piece of original work, Cullen was a great teacher. He published a work on nosology, in which he attempted to classify diseases by certain characteristics, somewhat after the manner in which Linnaeus had classified plants. Joseph Black (1728–1799), who taught chemistry, is especially famous as the discoverer that the gas, carbon dioxide, was given off in both respiration and combustion. Last, but by no means least, anatomy was taught by the Monros. Everyone is familiar with the dynasty of the Monro family in the chair of anatomy in the University of Edinburgh. For three generations, and they were long ones, extending over the period from 1720 to 1846, one hundred and twenty-six years, the chair of anatomy was occupied successively by Monros. They were designated by the ordinals, *primus, secundus* and *tertius*. Alexander Monro, *primus* (1697–1767), held the chair from 1720 until his resignation in 1758, when he was succeeded by his son, Alexander Monro, *secundus* (1733–1817). Both of these men were excellent teachers, and Alexander Monro, *secundus*, made many original contributions to anatomy and comparative anatomy. I shall only refer here to his discovery of the communication between

the lateral and third ventricles of the brain, which bears his name, the foramen of Monro. When Monro, *secundus*, was sixty-five years old, in 1798, he asked to have his son, Alexander Monro, *tertius* (1773–1856), appointed as his adjunct professor. The latter was only twenty-five years old and had as yet not given any token of particular fitness for the position. His father's request was, however, unfortunately acceded to. When Monro, *secundus*, retired, in 1808, the son was given the full professorship. He was a very stupid man, entirely unfit for the position. His lectures were so dull that Charles Darwin, who had begun the study of medicine at Edinburgh, gave it up rather than continue working under him. With his resignation, in 1846, the dynasty of the Monros was ended.

The Edinburgh degree of M.D. was held in much higher esteem than that obtainable anywhere else in the United Kingdom, ranking with that of Leyden, from which university its methods of teaching and practice were largely derived.

It is unfortunate that we cannot ascertain all the names of the young Americans who studied at Edinburgh, nor even their exact number. Many of them, like Ephraim McDowell, studied there, but did not take all the courses or remain long enough to get the degree—in McDowell's, and probably in many other instances, for financial reasons. Dr. J. Gordon Wilson,[1] of Chicago, has recently studied the records of the University of Edinburgh. He states that: "Edinburgh has on its roll of M.D.s fifteen names prior to 1765, representatives from almost all the American Colonies. From 1765 to 1779, despite the interruption of the American War of Independence, 1775–1783, there are one hundred and twelve names of Americans who received the degree of M.D. We have, however, good reason to believe that they represent only part of the greater number who studied there." Dr. John D. Comrie[2] states that "Out of 13 graduates in 1765, 5 belonged to Scotland, 5 were Americans, 2 English and 1 Irish."

An important feature in the life of many of the young Americans who studied at Edinburgh was the Medical Society of Edinburgh. This originated in 1734, when a group of six undergraduate medical students, who had been in the habit of spending social evenings at a tavern, agreed to meet once a fortnight at their respective

[1] President's Address to the Institute of Medicine of Chicago, December 4, 1928. Reprinted from the proceedings of the Institute of Medicine of Chicago, February, 1929, vol. vii.

[2] History of Scottish Medicine to 1860, London, 1927.

lodgings, upon which occasion one of their number should read a paper in rotation, which should be discussed by the others. In the following year all but one of the little group had left the University, but John Cleghorn, the one remaining, gathered to himself some other young men of the new arrivals at Edinburgh, among them William Cullen and John Fothergill, and continued the meetings. Two years later they increased their numbers and organized themselves into the Medical Society of Edinburgh. The Society met in a tavern until 1763, when it was granted permission to hold its meetings in a room at the Royal Infirmary. About the same time it agreed to form a library, which in 1778 contained no less than 1500 volumes. In 1776 it had prospered to such an extent that it was able to build a hall for itself. It had proved so useful and beneficial an organization as to receive the warm endorsement and support of the faculty and profession throughout Edinburgh. In 1779 it was granted a royal charter, and thus became the Royal Medical Society of Edinburgh. The Society opened its ranks to many of the young American students and was invaluable to them in forming connections with the best elements in the profession and in affording them the means of enjoying pleasant social intercourse with their fellow-students. In its hospitable bosom many friendships were formed which lasted long years after the sea had separated the friends. Several Americans—notably Rush, Physick and Caspar Wistar—were elected officers of the Society while in Edinburgh.

Having glanced at the advantages presented to the medical student in London and Edinburgh, let us now return to London for a few moments and consider some especial circumstances which prevailed at the time and had a most important bearing on the relations existing between the medical profession in Great Britain and the Colonies.

During the eighteenth century the Test Act, which required all students at the universities of Oxford and Cambridge to take the oath of allegiance to the Crown and the Church of England, excluded Quakers from studying at those institutions. Owing to their religious opinions, they were equally barred from pursuing a career in the army or in the church, and they were not eligible for election to Parliament. Consequently the English Quakers devoted themselves to financial or mercantile pursuits, in which many of them acquired large fortunes. With the expansion of intellectual activity

and scientific inquiry which marked the epoch, many Quakers sought an outlet for their mental activities in the study of medicine and the natural sciences. The English universities being closed to them, they either went to Edinburgh, where there were no such restrictions, or on the Continent, especially to Leyden, where Francis de la Böe and the great Boerhaave had developed a world-famed center of medical education. To two of these Quaker physicians in London, John Fothergill and John Coakley Lettsom, American medicine owes a great debt.

John Fothergill (1712–1780) was the son of a Quaker preacher. Probably his interest in America was largely due to the fact that his father had felt called upon to travel through the American Colonies on several preaching missions, and his brother Samuel had also visited them for the same purpose and had been much interested in the affairs of the Quakers in America. After graduating in medicine from Edinburgh, in 1736, John Fothergill went to London, and after serving several years at St. Thomas's Hospital, settled in practice in that city. He soon acquired one of the best practices in London. He said to Samuel Bard that he "had crept over the backs of the poor into the pockets of the rich." He was deeply interested in botany and the natural sciences. At his country seat, at Upton, he had a botanic garden in which the collection of foreign plants was only surpassed in England by that at Kew. One of his chief assistants in the formation of this collection was Peter Collinson (1699–1768), the English botanist, who was a correspondent of John Bartram (1699–1777), the Philadelphia botanist, with whom he arranged to send him living specimens of American trees and plants which Collinson in turn distributed to Fothergill and other English collectors. Collinson was also a correspondent of Benjamin Franklin and sent to the latter some electrical appliances with which Franklin conducted his famous experiments. When Franklin reported the results of these to Collinson, the latter showed his letters to Fothergill, who was so impressed that he communicated them to the Royal Society, by which they were so much esteemed that it bestowed the Copley Medal on Franklin in 1753. Fothergill caused Franklin's experiments to be published in London, and the two men maintained an active correspondence with one another until Franklin's arrival in London, 1757, when he and Fothergill became close personal friends, a friendship which was only terminated by death. During Franklin's long sojourns in England

every young colonist who went abroad to complete his medical studies sought his friendly advice and offices. Through his connection with Fothergill, Franklin was able to help them to obtain positions as dressers or assistants in the London hospitals, or get access to the special courses given by the Hunters or others of the great London teachers, and finally send them off to get their degrees at Edinburgh, Fothergill's alma mater. Fothergill aided the cause of medical education even more directly. To him belongs the credit of founding the first public medical library in the British colonies. In 1762 he sent to the managers of the Pennsylvania Hospital a book entitled *An Experimental History of the Materia Medica*, by William Lewis, F.R.S., which Fothergill wrote was a present to the hospital "for the benefit of the Young Students in Physic who may attend under the Direction of the Physicians." Shortly afterward, in 1763, the medical staff informed the managers that they proposed to devote all the fees to which they were entitled from students attending their courses at the hospitals to the purpose of buying books and establishing a medical library in the hospital. The managers approved the idea and agreed to furnish a room for the books. Dr. Fothergill's present is still on its shelves. I shall refer again to another gift of Dr. Fothergill's to the Pennsylvania Hospital, a beautiful set of large anatomical pictures to be used in teaching anatomy. They were made by van Rymsdyk, a Dutch artist living in London, and cost Dr. Fothergill the handsome sum of two hundred guineas. Van Rymsdyk was the best anatomical artist of his day. He made most of the illustrations for William Hunter's famous book on *The Gravid Uterus*. In honor and remembrance of Dr. Fothergill, the Medical Society of London established a Fothergillian medal. In 1888 this medal was awarded to our late distinguished president, Dr. Hobart A. Hare, for an essay on *Diseases of the Mediastinum* and was bequeathed by him to the College of Physicians, where it is now deposited.

John Coakley Lettsom (1744–1815) was Fothergill's close friend and colleague. He is probably chiefly recalled to most of us by the famous couplet on his name which is current in many versions:

> "When patients sick to me apply,
> I physicks, bleeds and sweats e'm.
> There's some get well and some that die,
> What's that to me,
> I.[1] Lettsom.

[1] The initial letter J was frequently written I at that time.

Lettsom was the son of English parents, living in the West Indies. He was one of the last of seven pairs of twins to which his mother gave birth, and he and his twin brother were the only ones to survive their infancy. Lettsom studied medicine in London, Paris and Edinburgh, but got his M.D. at Leyden in 1769. In July, 1800, he sent over some of Dr. Jenner's vaccine material to Dr. Benjamin Waterhouse in Boston, which is generally supposed to have been the first utilized in this country for the purpose of vaccination. Lettsom was very zealous in befriending American students in London, and acted as agent in England in the purchase of books for the Pennsylvania Hospital's library.

That the memory of these two early benefactors of American medicine was kept green by at least some who appreciated their efforts, we gather from the fact that Dr. Benjamin Rush's son, James Rush, named two tracts of land which he owned in western Pennsylvania after them: one on Sugar Creek was called Lettsom, and the other on Loyalist Creek, Fothergill.

Another kind friend to the American students in London was Sir John Pringle (1707–1782), the famous pioneer military hygienist, at one time surgeon-general of the British Army. He used to make them welcome to his house, as well as aiding them with advice as to their studies.

It was the ever-helpful Benjamin Franklin, however, who was the most important aid to his young fellow-countrymen. It may be recalled that Franklin visited England three times. His first trip was made in 1725–1726, when, at the instigation of Sir William Keith, he went over to London, where he worked at his trade as a printer. In 1757 he went to London as the representative of Pennsylvania in the disputes between the inhabitants of that province and the proprietaries, the Penns. On this occasion he stayed in England for six years, returning to Philadelphia late in 1763. During this sojourn he frequented the best circles in London, was a zealous attendant at the meetings of the Royal Society and was treated with much consideration by Fothergill, Lettsom and many other prominent persons. He made a visit to Scotland, where he was much entertained and made many friends, among them Lord Kames and Sir Alexander Dick. The University of Aberdeen conferred the honorary degree of LL.D. upon him in 1759. Franklin's last visit to London began in 1764, when he was sent over as agent for Pennsylvania, New York and New Jersey in the disputes then pending with the government of Great Britain. He was received

with open arms by his English friends. This time he remained for nine long years, not returning to Philadelphia until 1775. The importance of these trans-Atlantic sojourns of Benjamin Franklin in relation to scientific, educational and medical affairs in the Colonies, especially in Pennsylvania, cannot be overestimated. Treated on terms of friendship and esteem by the most intellectual circles in London, Franklin used his opportunities to aid his countrymen in their efforts to better cultural affairs in America to the fullest extent, and that they amply repaid him, at least as far as the profession of medicine was concerned, I hope to demonstrate. There was one noticeable exception to the favor accorded Franklin by the London intellectuals. Although our fellow-townsman, A. Edward Newton, has recently published a most delightful report of an imaginary dinner at the publisher Strahan's at which both Samuel Johnson and Benjamin Franklin were guests, he admits that he doubts if they ever encountered one another. There is no mention of any such meeting in Boswell. Perhaps it was just as well when we recall that on one occasion, when dining with Dilly, the publisher, Johnson suddenly burst forth with: "I am willing to love all mankind except an American," to the astonishment of Miss Seward, the poetess, and Mrs. Knowles, a gentle Quakeress, who were among the guests.

Franklin's own views on the educational opportunities for the student of medicine in London and Edinburgh are given in a latter to Dr. Thomas Bond, written February 5, 1772.[1]

[1] The letter is reproduced by Dr. William Pepper in his most excellent series of articles on The Medical Side of Benjamin Franklin, which were published in the University of Pennsylvania Medical Bulletin in 1910: "I suppose your son Richard will spend some time in London, where, by what I have heard, Physic and Surgery may be studied to as great advantage as in any Part of the World, by Attending the Anatomical Lectures and Hospitals, conversing with the most eminent Practitioners, and Reading under their Advice and Direction; and yet the general run is at present to Edinburgh; there being at the Opening of the Schools, when I was there in November last, a much greater number of medical Students than had ever been known before. They have indeed a Set of Able Professors in the several Branches, if common Opinion may be rely'd on. I who am no Judge in that Science, can only say that I found them very sensible Men, and agreeable Companions. I will endeavor to obtain Sir John Pringle's Advice in the Affair, as you desire. Every Wednesday evening he admits young Physicians and Surgeons to a Conversation at his House, which is thought very improving to them. I will endeavor to introduce your Son there when he comes to London. And to tell you frankly my Opinion, I suspect there is more valuable knowledge in Physic to be learnt from the honest candid Observations of an old Practitioner, who is past all desire of more Business, having made his Fortune, who has none of the Professional Interest in keeping up a Parade of Science to draw Pupils, and who by Experience had discovered the inefficacy of most Remedies and Modes of Practice, than from all the formal Lectures of all the Universities upon Earth. I like therefore a Physician's breeding his Son to Medicine, and wish the Art to be continued with the Race, as thinking that must be upon the whole most for the Publick Welfare."

And now having considered the inducements which led young Americans to go to London and Edinburgh for medical study, and what opportunities were afforded them when they got there, let us come back to Philadelphia and see what use they made of these opportunities after their return. The biographies of these men who laid the foundation of medicine in this city have been so fully written and are so readily available that I shall not give them in detail, but shall only deal with that part of their careers which bears upon our subject, or which throws some light upon their personal characteristics, because I cannot but think that the latter were much affected by their intercourse with the cultivated members of their profession with whom they came in contact in London and Edinburgh. The first Philadelphian to utilize the advantages of his studies in London for the benefit of his fellow-practitioners in Philadelphia was Thomas Cadwalader (1708–1779). He went abroad and worked under the great Cheselden. After his return to Philadelphia, about 1750, he gave some anatomical demonstrations to his colleagues which were greatly appreciated. Dr. Cadwalader was one of the consulting physicians to the Pennsylvania Hospital. He wrote *An Essay on the West India Dry Gripes*, which was printed by Benjamin Franklin. The "Dry Gripes" was very prevalent in Philadelphia because of the very general habit of the citizens of drinking punch made from Jamaica rum. The latter was distilled in leaden pipes and the disorder was really lead poisoning. Dr. Cadwalader was distinguished for his benevolence and his kind and gentle manners. The latter saved his life on one occasion. A Lieutenant Bruhman, of the British Army, became suddenly insane and sallied forth with a gun resolved to shoot the first man he should meet. This happened to be Dr. Cadwalader, who, bowing pleasantly, said to him: "Good morning, Sir, what sport?" The lieutenant replied civily and fell into conversation, during which he was so charmed with the doctor's pleasing ways that he abandoned his murderous intentions. They parted, and a few minutes later the lieutenant met Mr. Robert Scull, whose manners failed to charm him as had those of Dr. Cadwalader; so he shot him dead, for which he was duly executed.[1]

A close associate and friend of Thomas Cadwalader was John Redman (1722–1806), the first President of this College, whose picture hangs back of our President's chair. Although he did not

[1] Watson: Annals of Philadelphia.

graduate from the University of Edinburgh, he studied there for a year, and also spent a year working at Guy's Hospital in London, before he went to Leyden, from which University he received his M.D. in 1748. He was one of the consulting physicians to the Pennsylvania Hospital, and had probably more private pupils, or apprentices, than any other physician of his day in Philadelphia. Although he never taught in the medical school, he was a trustee of the College of Philadelphia and took a very active part in its affairs. It was said of him that—"In a sick room he possessed the virtues and talents of a specific kind; he suspended pain by his soothing manner, or chased it away by his conversation, which was occasionally facetious and full of anecdotes, or serious and instructive, according to the nature of the patient's diseases or the state of their minds." We have the following word picture of Dr. Redman in his old age, which Watson,[1] quotes from one who had often seen him: "The doctor, who lived in Second Street near Arch, had retired from practice altogether, and was known to the public eye as an antiquated-looking old gentleman usually habited in a broad-skirted dark coat, with long pocket flaps buttoned across his under dress; wearing, in strict conformity with the cut of his coat, a pair of Baron Steuben's military shaped boots, coming above the knees for riding; his hat flapped before and cocked up smartly behind, covering a full-bottomed powdered wig, in the front of which might be seen an eagle-pointed nose separating a pair of eagle-black eyes; his lips exhibiting now and then a quick motion, as though at the moment he was endeavoring to extract the essence of a small quid. As thus described in habit and person, he was to be seen almost daily, in fair weather, mounted on a short, fat, black, switch-tailed horse, and riding for his amusement and exercise, in a brisk racking canter about the streets and suburbs of the city."

And now let us consider the men who organized and taught in the first medical school founded in this country, the Medical School of the College of Philadelphia, as the University of Pennsylvania was then known. At the time when the medical school was founded, in 1765, there were five physicians on the board of trustees of the College, viz., Thomas Bond, Phineas Bond, Thomas Cadwalader, William Shippen, Sr., and John Redman, all of whom with the exception of William Shippen, Sr., had enjoyed the advantage of studying abroad, and the first six men who held professorships—John Morgan,

[1] Annals of Philadelphia.

William Shippen, Jr., Adam Kuhn, Benjamin Rush, Philip Syng Physick and Caspar Wistar—had all of them received the degree of M.D. from the University of Edinburgh. This furnishes a ready explanation for the fact mentioned by Carson,[1] in his history of the Medical Department of the University of Pennsylvania: "From a comparison of the course of instruction in the College of Philadelphia, from the time of its inception to that of its complete organization, with that of the University of Edinburgh, there can be no doubt that this distinguished school was taken as the model for imitation." Until the temporary abrogation of the charter of the College of Philadelphia, in 1779, every member of the faculty was a graduate in medicine of the University of Edinburgh.

A brief review of the work of the teachers in the Medical School of the College of Philadelphia during its early years will show how splendidly the leaven of London and Edinburgh worked in the growth of the young school.

To John Morgan (1735–1789) is ascribed the honor of having been the founder of the Medical Department of the University of Pennsylvania. After serving an apprenticeship with Dr. John Redman, Morgan went to London in 1760, where he worked chiefly with William Hunter.[2] Benjamin Franklin, who was residing in London as agent for Pennsylvania, showed him great kindness, introducing him to Fothergill and many other prominent persons. When in the following year Morgan went to Edinburgh, Franklin gave him letters of introduction to Lord Kames and Dr. William Cullen. With the latter Morgan became on very close terms. He talked to him about his plan to found a medical college in Philadelphia, and Cullen probably gave him much valuable advice. Morgan got his M.D. from the University of Edinburgh in 1763,[3] and then went to Paris, and later took quite an extensive Continental tour, in the course of which he visited Voltaire at Ferney and had a most interesting interview with that philosopher. Morgan also visited the famous Italian physician, Morgagni, at Bologna, and the latter

[1] A History of the Medical Department of the University of Pennsylvania, Philadelphia, 1869.

[2] John Hunter received his commission as surgeon in the Army in 1759, and must have been absent on the expedition to Belle Isle during much of the period of John Morgan's stay in London, or if he had returned he had not resumed teaching.

[3] Morgan's thesis was entitled, *De Puopoisei*. In it he maintained that pus was a secretion from the inner walls of the bloodvessels, a view subsequently put forth by John Hunter and usually spoken of as original with him. There is no doubt, however, of Morgan's priority, though Hunter was not aware of Morgan's views when he published his own.

presented him with a copy of his great work on pathological anatomy *De Sedibus Morbis.*[1] During his stay abroad Morgan was the recipient of many honors. He was made a fellow of the Royal Society, member of the Belles Lettres Society of Rome, associate of the Academie Royal de Chirurgie of Paris and a licentiate of the Royal Colleges of Physicians of both London and Edinburgh.[2] Morgan returned to Philadelphia early in 1765, and at the outset of his career as a practitioner introduced several striking departures from the proceedings in vogue among the physicians of the city. He announced that he had brought over from England a man named David Leighton, who was qualified to practise surgery and pharmacy, and that he would turn over to him all the work of that nature which might be necessary in the course of his practice; or, if his patients wished, they might employ an apothecary or surgeon of their own choice, but that he himself would not dispense medicines, nor do any surgery, and that he also expected to receive his fee at each visit, according to the custom in London. These innovations did not meet the approval of his professional colleagues, and it was not until Dr. Abraham Chovet and, later, Dr. John Jones followed his example as regarded dispensing medicines that it was generally adopted. Another innovation which Morgan is said to have introduced into Philadelphia was the practice of carrying an umbrella. The Reverend Dr. Duche and Dr. Chancellor joined him in appearing on the streets armed with this curious appliance. "Before this some doctors and ministers used an oiled linen cape hooked around their shoulders, looking not unlike the big capecoats now in use, and then called a roquelaure. It was only used for very severe storms."[3] The first umbrellas were made of oiled cloth laid over heavy rattan ribs.

While abroad, Morgan had been carefully working over the project of organizing a medical school in connection with the College of

[1] This book was presented to the College of Physicians of Philadelphia by Morgan. It bears the following inscription written by Morgagni on the title-page of each volume. Inscription in Volume 1: "Viro Experientissimo et Humanissimo, D. D. Joanni Morgan, Auctor." For the second Volume: "Viro de Re Anatomia bene merito D. D. Joanni Morgan, Auctor." As will be seen, there is nothing in this inscription to bear out the oft-repeated assertion that Morgagni wrote a punning Latin inscription approximating Morgan's name to his own, and addressing him as his son.

[2] Morgan's journal, which he kept during his travels on the Continent, was printed for private circulation by Colonel A. S. Morgan, in 1912. It is a most interesting little book.

[3] Watson's Annals of Philadelphia.

Philadelphia, and he submitted his plan to the trustees on May 3, 1765. It was unanimously approved by the Board and Morgan was elected to the professorship of the theory and practice of physic. At the commencement exercises on May 30, 1765, he pronounced his famous "Discourse upon the Institution of Medical Schools in America." Morgan had composed "The Discourse" in London and had submitted it to John Fothergill and William Hunter for their criticisms and suggestions. In it he presents the necessity for a medical school and the advantages to be derived from it, with an outline of the plan for its organization. The "Discourse" was a splendid piece of work and won universal commendation. It shows how thoroughly Morgan was imbued with the Edinburgh tradition and that he planned to place the newly-founded medical school on the highest plane. In September, 1765, the trustees appointed William Shippen, Jr., to the professorship of surgery and midwifery in the school, and a little later Kuhn and Rush were added to the faculty.

Things went splendidly until along came the Revolution. Congress appointed Morgan Director-General of the Military Hospitals and Physician-in-Chief to the American Army. Shippen also went into the service, and Benjamin Rush was one of the Signers, and very active in the affairs of Congress, as well as later serving in the Army. I shall not go into details of the public services of any of these men. Suffice it to say that, although Morgan did splendid work under the greatest difficulties, he made enemies by his attempts to introduce proper discipline into the medical department and correct abuses. In January, 1777, he was summarily dismissed from his position by Congress. He demanded an inquiry and wrote his noble "Vindication" of his conduct. Two years later, a Congressional Committee was appointed to investigate the matter, and its report completely exonerated Morgan. But Morgan never recovered from the blow to his pride and self-esteem. He withdrew from teaching and practice and lived in retirement until his death at the comparatively early age of fifty-three, on October 15, 1789.

Benjamin Rush wrote of Morgan: "He possessed an uncommon capacity for acquiring knowledge. His memory was extensive and accurate; he was intimately acquainted with the Greek and Latin classics; had read much in medicine, and in all his pursuits he was persevering and indefatigable. He was capable of friendship, and in his intercourse with his patients discovered the most amiable

and exemplary tenderness. I never knew a person who was attended by him that did not speak of him with gratitude and respect." Morgan was elected physician to the Pennsylvania Hospital in 1773, but he resigned in 1783, owing to a difference of opinion with the Board of Managers over the charging of fees to patients suffering from venereal disease who were sent to the Pennsylvania Hospital from the Philadelphia Almshouse.

Morgan's career ended in bitterness and disappointment, which is all the more sad by contrast with its brilliant beginning. His only writings of note were his *Discourse on the Founding of Medical Schools* and his *Vindication*. Curiously enough, he received a prize some years before the Revolution for an essay *On the Reciprocal Advantages of a Permanent Union Between Great Britain and the American Colonies*.

The second man appointed to a professorship in the medical school of the College of Philadelphia was William Shippen, Jr. (1736–1808). After graduating from the College of New Jersey (Princeton), he began the study of medicine with his father, one of the best-known physicians of his time in Philadelphia. In 1757 the latter sent him abroad to complete his studies. Watson[1] quotes from a letter written by the elder Shippen to an English correspondent, which states so well his reasons for sending his son abroad to study, and lays down such a typical course for him to pursue that I shall quote it. Doubtless the father of many a young man who was sent abroad to finish his education wrote or thought as did Dr. Shippen, Sr.: "My son has had his education in the best college in this part of the country, and has been studying physic with me, besides which he has had the opportunity of seeing the practice of every gentleman of note in our city. But for the want of that variety of operations and those frequent dissections which are common in older countries, I must send him to Europe. His scheme is to gain all the knowledge he can in anatomy, physic and surgery. He will stay in London for the winter and shall attend Mr. Hunter's anatomical lectures and private dissections, injections, etc., and at the same time go through a course of midwifery with Dr. Smellie; also enter as a pupil in Guy's Hospital. As soon as the season is over, he may go to France and live with Dr. Leese in Rouen, and there study physic until he can pass an examination and take a degree." Young Shippen went to London accordingly, where he studied anatomy

[1] Annals of Philadelphia.

and surgery under William and John Hunter and George McKenzie. He associated much with William Hewson (1739–1774), William Hunter's brilliant young assistant, who made some most remarkable discoveries regarding the lymphatics and the phenomena of the coagulation of the blood, before his premature death at the early age of thirty-five years. Hewson married the daughter of Benjamin Franklin's London landlady, Mrs. Stevenson, and it may be recalled that after his death his widow brought her children over to Philadelphia to live. Perhaps Shippen's friendship for her husband prompted her to take this step, as much as Benjamin Franklin's known advice to her to do so. Shippen went from London to Edinburgh, where he got his M.D. in 1761.[1] Some years later, in 1786, he was elected a fellow of the Royal College of Physicians of Edinburgh. Before returning to Philadelphia, Shippen travelled in France. As Great Britain was then at war with France, he could not have done this had it not been for the good offices of Sir John Pringle. It appears that there was a wealthy British female who suffered from tuberculosis and wished to go to the south of France. George III personally solicited permission from the French government for her to do so, and they with Gallic chivalry acceded to his request. Pringle secured Shippen the job of going along as her traveling physician, and in that capacity he had an opportunity of visiting the great French medical school at Montpellier and much else of interest.

Shippen got back to Philadelphia in 1762. While in London he had talked much to Dr. John Fothergill about his plans for giving courses in anatomy and midwifery in his native city, and that generous benefactor to American medicine had been so deeply interested that he had sent over in his care to the Pennsylvania Hospital the set of anatomical drawings and casts to which I have already referred, stating in a letter to the Board of Managers that he understood the difficulty of procuring bodies for dissection: "In the want of real subjects these will have their Use & I have recommended it to Dr. Shippen to give a Course of Anatomical Lectures to such as may attend. He is very well qualified for the subject & will soon be followed by an able assistant, Dr. Morgan, both of whom I apprehend will not only be useful to the Province in their Employment, but if suitably countenanced by the Legislature will be able to erect a School of Physic amongst you." Although Dr. Shippen

[1] Shippen's thesis was entitled, *De Placentæ cum Utero Nexu.*

was not elected to the staff of the hospital until 1778, he was allowed the use of the Fothergill pictures and casts in connection with the private teaching courses which he organized, and he used to be present at the hospital on certain days in order to "explain and demonstrate to such persons who are willing to give a dollar each for the benefit of the hospital."

In the autumn of 1762 Shippen began giving systematic courses in anatomy, to which some time later he added midwifery. These courses were the first successful effort to give systematic instruction in these subjects in America, and Shippen continued to give them after his appointment as professor in the medical school. This was directly in line with the Edinburgh policy of extramural schools, that is, private schools conducted outside of the University curriculum. During the Revolution, Shippen's courses were necessarily interrupted. After his resignation from the army, Shippen devoted himself to his teaching with renewed ardor and the greatest success. Caspar Wistar, in his "*Eulogium*" *of Shippen*, states that he had as many as four-hundred students in his anatomy class at one time.[1] In 1779, by an act of legislature, the College of Philadelphia was deprived of its charter and privileges, which were arbitrarily transferred to a new institution created by the legislature with the title of the University of the State of Pennsylvania. Shippen was appointed to the professorship of anatomy, surgery and midwifery in the new school. Ten years later, in 1789, another act of legislature corrected the injustice by restoring its charter and rights to the College of Philadelphia. For several years the two institutions existed, but it was obvious that there was no reason for such a duplication. Accordingly, in 1791, the College of Philadelphia was united with the University of the State of Pennsylvania, to form the University of Pennsylvania. Shippen continued to hold the chair of anatomy, surgery and midwifery in the new institution, with Caspar Wistar, also a graduate of the University of Edinburgh, as his adjunct professor. In 1798 Shippen sustained a great loss by the death of his son, a young man of great promise, who had, however, been in ill-health for a number of years. According to his colleague and biographer, Caspar Wistar, Shippen never recovered from this blow: "It cut the sinews of his exertions and left him gradually to wither, the amiable victim of paternal affection. From this time

[1] Dr. G. W. Norris, Early History of Medicine in Philadelphia, gives the maximum number of students as two hundred.

his business as a practitioner declined. He seldom lectured on anatomy, and generally with reluctance, though when he did lecture he always gave the greatest pleasure to his class." In 1805 Shippen was elected President of the College of Physicians, succeeding John Redman, and the same year he gave up teaching surgery, retaining the chair of midwifery and anatomy until his death on July 11, 1808. Wistar gives a charming little sketch of Shippen's personality when in his prime: "Nature had been uncommonly bountiful in the form and endowments of Dr. Shippen. His person was graceful, his manners polished, his conversation various, and the tones of his voice singularly sweet and conciliatory. In his intercourse with society he was gay without levity and dignified without haughtiness or austerity. He belonged to a family which was proverbial for good temper. His father, whom he strongly resembled in this respect, during the long life of ninety years, had scarcely ever been seen out of humor. He was particularly agreeable to young people. Known as he was to almost every citizen of Philadelphia, it is probable there was no one who did not wish him well."

The third professor appointed to the medical faculty of the College of Philadelphia was Adam Kuhn (1741–1817). He began his medical studies under his father, who was a physician, and in 1761 went abroad to continue them. Kuhn varied the usual proceedings somewhat by going first to the University of Upsala, Sweden, where he studied especially under the famous botanist, Linnæus. In 1764 he went to London, where he remained a year before going to Edinburgh, from which University he received his M.D. on June 12, 1767.[1] Before his return he had travelled also in France, Holland and Germany. Upon his arrival in Philadelphia, in January, 1768, he was appointed professor of materia medica and botany in the medical school of the College of Philadelphia. In 1779 he resigned his chair to become professor of the theory and practice of medicine in the University of the State of Pennsylvania and, after the union of that institution with the College of Philadelphia to form the University of Pennsylvania, he occupied the same chair in the University until his resignation in 1797. Dr. Kuhn was physician to the Pennsylvania Hospital from 1775 to 1798 and was the third President of the College of Physicians, having been elected to succeed William Shippen, Jr., in 1808. His health failed to such a

[1] His thesis, *De Lavatione Frigida*, was dedicated to Linnæus.

degree that he retired from practice in 1815. Two years later he died on July 5, 1817. Dr. Kuhn contributed but little to medical literature. Besides his thesis, his only other publication was a short letter addressed to Dr. John Coakley Lettsom on the diseases following transplantation of the teeth, which was published in the first volume of the *Memoirs of the Medical Society of London*. Of his characteristics we gather from notices written by contemporaries, that though reserved in his common intercourse with mankind, "in the company of his friends no man was more affable and communicative. . . . His kind and unassuming behavior to younger physicians, his manners void of ostentation and his firmness and decision of conduct will long be remembered. He was distinguished for his extreme devotion to punctuality." Dr. Kuhn was not remarkable for the powers of imagination, but in sound judgment he greatly excelled. His talent for observation was profound. He was through life a studious reader, a lover of music from his youth, remarkably abstemious and regular in his diet and neat in his person."[1]

The sour Charles Caldwell hated Kuhn, as he did most of his colleagues in Philadelphia. In his *Autobiography* Caldwell writes of Kuhn: "He was by far the most highly and minutely furnished specimen of old-school medical production I have ever beheld. His hair, unusually coarse and strong, of which nature had furnished him with an exuberant abundance and given to it so trim and permanent a setting that, at the age of seventy-five, he possessed, perhaps, as much of it as he did at that of twenty-five: that appendage of the head his hairdresser so arranged as to give it the resemblance of a fashionable wig, well pomatumed, stiffly curled and richly powdered. His hand- and bosom-ruffles were full and flowing, his breeches were black, his long-skirted waistcoat white or buff and his coat snuff-colored. In his hand he carried a gold-headed cane, in his waistcoat pocket a gold snuff-box, and his knee- and shoe-buckles were of the same metal. . . . He entered each sick-room at a given time, spent a given number of minutes in examining and prescribing and never suffered the slightest deviation to be made from his directions. Weak sage-tea was a favorite beverage which he often prescribed as a drink for the sick. If, as was sometimes the case, the kind nurse or attendant took the liberty of saying to

[1] Thacher, in his American Medical Biography, quotes most of the above account of Kuhn from the Eclectic Repository, vol. viii.

him as he was moving toward the door to make his exit from the room: 'Doctor, should the patient want to drink a little toast-water or lemonade, I suppose I may give it to him?' He would turn and reply, with oracular solemnity and decisiveness: 'I have directed (or I have said) weak sage-tea. Good morning, madam.'" His lectures, Caldwell charges, were merely a rehash of those of Cullen. This bitter criticism was written in spite of the fact that when Caldwell had been very ill he sought Kuhn's professional services and had been visited by him thrice daily and he had to acknowledge his skill and success as a practitioner.

One cannot hope to do justice to such a great man as Benjamin Rush (1745–1813), the fourth man elected to a professorship in the Medical School of the College of Philadelphia, in a thumbnail sketch. His achievements were too many and too various. The great pity is that, as yet, there has been no adequate biography written of this great physician, educator, scholar and statesman. After graduating from Princeton, Rush served an apprenticeship with Dr. John Redman and was one of the students who attended Shippen's first course on anatomy. His apprenticeship finished, he went abroad and, on June 1, 1768, received his M.D. from the University of Edinburgh.[1] I am indebted to Dr. J. Gordon Wilson, of Chicago, for directing my attention to an interesting correspondence in *Notes and Queries*, volumes ix and x, 1854. It quotes the following from the Records of the Town Council of Edinburgh: "March 4, 1767. The Council admits and receives Richard Stocktoun (sic), Esq., of New Jersey, and Benjamin Rush, Esq., of Philadelphia, to be burgesses and gild brothers of this city in the most ample form." No reason is given why such an honor should have been conferred on the two young Americans.[2]

[1] Rush's thesis, *De Coctione Ciborum in Ventriculo*, was especially remarked for the elegance of the Latin in which it was written.

[2] Notes and Queries also contains the following: Benjamin Rush. In answer to Inquirer, I am tempted to send the following extract from a recently discovered letter from Dr. Rush to a friend in Philadelphia, describing a very peculiar method of presenting the freedom of the city to strangers which prevailed in Edinburgh ninety years ago and to which allusion is made by some of the English novelists of the last century. I have heard that the usage prevails to this day in Rome, Naples and other Italian cities. The letter from which I quote is dated December 29, 1784:

Edinburgh is built upon one-third less ground than Philadelphia, but contains double the number of inhabitants. I think they compute eighty thousand souls in the city. The reason why they occupy so much less room is owing to the height of their houses, in each of which seven or eight families reside. There is one common pair of steps which communicate with all the rooms of the house. These steps are open and exposed, and are trod by everybody in the same manner as the public

The winter following his graduation was spent in London, with a sojourn in Paris. Returning to Philadelphia in 1769, he was elected professor of chemistry in the College of Philadelphia. When Dr. Kuhn resigned from the College, Dr. Rush succeeded him as professor of the theory and practice of medicine. After the formation of the University of Pennsylvania he was elected professor of the institutes of medicine and of clinical medicine in the new University. He was elected one of the physicians to the Pennsylvania Hospital in 1783, and he fulfilled his duties to the Hospital and the University with the most absolute regularity until his death in 1813. It should be remembered that he was a Signer of the Declaration of Independence a very active member of the Continental Congress and during the Revolution he served as physician general in the Continental Army. From 1799 to 1813 he was treasurer of the mint of the United States. He was active in many benevolent and other public movements, particularly that for the abolition of slavery, temperance reform and the abolition of capital punishment. He was the chief factor in the founding of Dickinson College and a book has been published recently devoted entirely to his services to education.[1]

I cannot refer more than very briefly to Rush's wonderful services during the epidemics of yellow fever which devastated Philadelphia in 1793, 1794, 1797, 1798 and 1803. He had witnessed a previous epidemic while a student in 1762. Throughout these terrible times he stayed at his post, saw more patients than any other physician in the city, and was more successful in the treatment of his cases. At first he believed the disease was contagious, but later he changed his views and held that it was not. He believed that it was of domestic origin and not imported by ships from Southern ports. In this

streets. Dr. Franklin called them, some years ago when in Scotland, upon this account, perpendicular streets. The inhabitants, although they live together in their human hives, are entire strangers to (one) another. There is a family living above me and another immediately below me, and yet I know no more of their names or persons than you do. This way of living subjects the inhabitants to many inconveniences, for, as they have no yards or cellars, they have of course no necessary houses and all their filth of every kind is thrown out of their windows. This is done in the night generally, and is carried away next morning by carts appointed for that purpose. Unhappy they who are obliged to walk out after ten or eleven o'clock at night. It is no uncommon thing to receive what Juvenal says he did, in his first satire, from a window in Rome. This is called here being *naturalized*. As yet I have happily escaped being made a freeman of the city in this way, but my unfortunate friend Potts has gained the honour before me.

[1] Benjamin Rush and his Services to Education. By Harry G. Good, Berne, Ind., 1915.

he differed from the opinion of the majority of the Fellows of the College of Physicians. This led to his resignation from this body and to the formation of the short-lived Philadelphia Academy of Medicine by those of his colleagues who adhered to his opinion. It is hard for us to realize the bitterness which was engendered in those days by differences of views on purely scientific or professional matters. Rush relied chiefly on copious bleeding and large doses of calomel in his treatment of yellow fever and he certainly was, as before said, more successful with his patients than most of his colleagues. They heaped ridicule on his methods. He used to give what he termed his "10-and-10" powder, containing 10 grains of calomel and 10 grains of jalap, frequently repeated, and he spoke of calomel as his "Samson" because of its power against yellow fever; but the wags said it should be called so because of the numbers it had slain. They also referred to him continually as Dr. Sangrado, after the famous bleeder in *Gil Blas*. Rush suffered an attack of yellow fever himself and attributed his recovery to the copious bleeding and large doses of calomel to which he was subjected.

Rush was the pioneer American alienist. He advocated the treatment of insanity as a disease and the management of its victims with humanity. He was able to put his enlightened views in practice at the Pennsylvania Hospital, where he developed an excellent service for the mentally disordered. His writings fill many volumes and contain much of permanent value. Rush was a profoundly religious man. He took an active part in founding the Bible Society and was a strong advocate of daily bible-reading in the public schools. His friend and eulogist, Dr. Francis, of New York, wrote: "Those who had the happiness to experience the delights of his conversation will long recollect with pleasure his unassuming modesty and the rich stores of knowledge he poured forth on the most instinctive topics, even when his opinions were solicited, not as the dictates or admonitions of a superior but as the kind advice of a friend and equal. He never evinced any of that haughtiness and affectation of importance which sometimes attach to men of eminence and which so materially lessens the pleasures and comforts of social life."

The four Edinburgh graduates of whose careers I have given you a brief outline made up the first full faculty of the medical school of the College of Philadelphia. In addition we have seen that, in accordance with the custom at Edinburgh, extramural courses

were given on anatomy and midwifery by William Shippen, Jr., and, besides these, extramural courses on clinical medicine were given at the Pennsylvania Hospital by Thomas Bond, who had studied at Edinburgh, although he did not remain there to receive his degree. The medical faculty as thus organized continued practically unchanged until the temporary disruption of the College of Philadelphia. Shortly after its reorganization as the University of Pennsylvania two other graduates of the University of Edinburgh became members of the faculty, who maintained the Edinburgh tradition with great effect for many years. These two men were Philip Syng Physick and Caspar Wistar.

Philip Syng Physick (1768–1837) has been justly honored with the title of "Father of American Surgery." He graduated from the collegiate department of the University of Pennsylvania in 1785. It is related by his son-in-law and biographer, Dr. Jacob Randolph, that it was probably due to his father's urging that young Physick undertook the study of medicine. One day Mr. Physick cut his finger very severely and his son showed so much aptitude in dressing the injury that he "determined in his mind to make him a surgeon." Randolph supplements this story with the curious statement: "I feel compelled to confess that I do not think Dr. Physick himself could support pain with the same degree of fortitude and composure which we have sometimes met with in persons who suffered to an equal extent with himself; it is undeniable, however, that he was extremely unwilling to be the source of inflicting pain upon others." Physick began the study of medicine under Dr. Adam Kuhn, and soon after manifested his susceptibility in this direction. Dr. Kuhn wished him to witness an amputation which was to be performed at the Pennsylvania Hospital, "but understanding perfectly well the peculiar temperament of his pupil, he gave it as his advice that his father should go with him, and fortunately too, inasmuch as his son became so sick during the operation that it was necessary to lead him from the amphitheatre before it was concluded." A curious beginning, indeed, for the career of a great surgeon. While studying with Kuhn, Physick also attended the medical lectures at the University of Pennsylvania, but did not try for a degree, as at the end of three years' study he sailed for England, accompanied by his father. They arrived in London in January, 1789. Young Physick was accepted by John Hunter as a house pupil, and for one year he worked hard at anatomy and surgery under the greatest

teacher of his time. He was a great favorite with Hunter, who, January 1, 1790, secured for him the position of house surgeon at St. George's Hospital. This much-coveted appointment is a token of the favorable impression the young man must have made on Hunter. There were many applicants and it was a great triumph to have received it. At the expiration of his year's term of service, in January, 1791, Physick received his diploma from the Corporation of Surgeons of London.

Hunter wished Physick to remain with him as his assistant, but Physick declined this flattering offer, although he lived in Hunter's house and helped him in his work from January until May, 1791, when he left London for Edinburgh. One year later, May, 1792, he received his M.D. from that University.[1] He remained in Edinburgh a few months, working at anatomy and surgery. In September, 1792, he returned to Philadelphia and started to practise. In the subsequent year, 1793, Philadelphia suffered from a terrible epidemic of yellow fever. Physick served as physician to the hospital at Bush Hill, and in that capacity showed the greatest skill and bravery, by which he won the lasting esteem and friendship of Stephen Girard. Physick suffered an attack of the disease himself and had it again in 1797. In 1798 he again acted as resident physician at Bush Hill Hospital. He was a devoted follower of Benjamin Rush, believing yellow fever to be of domestic origin and noncontagious. As has been mentioned, Rush resigned from the College of Physicians of Philadelphia because that body had issued a statement in which it was maintained that yellow fever was imported into the city by ships and not of domestic origin. Because of his devotion to Rush and adherence to his views, Physick never became a Fellow of the College.

Physick was elected to the staff of the Pennsylvania Hospital in 1794 and served until 1816. In 1800, at the request of a number of medical students at the University of Pennsylvania, Physick began giving private lectures on surgery. Six years later (1806) he was elected professor of surgery in the University. This chair he held until 1819, when he was transferred to that of anatomy, which he resigned in 1831. Dr. Randolph says: "Dr. Physick's manner as a public lecturer was grave, dignified and impressive to an extraordinary degree. His style was clear and comprehensive, simple yet chaste. He was uniformly careful never to say too much.

[1] His thesis, *De Apoplexia*, was dedicated to John Hunter.

His choice of language was remarkably good, and he possessed the happy faculty of communicating knowledge agreeably and well in as great perfection as any other man I have ever heard lecture. . . . His lectures were all carefully prepared and written out." Physick never wrote a book and made but few contributions to medical literature, but those of great importance. He was president of the Phrenological Society of Philadelphia in 1822.

Some of Dr. Physick's most important work may be briefly noticed. Physick was especially expert in the treatment of fractures and dislocations, devising a number of very useful splints and dressings for them. In ununited fractures he achieved much success by using a seton passed between the fragments. He devised a curved bistoury for use in operations for fistula in ano, and originated an operation for the cure of artificial anus which was much superior to any heretofore known. In the *Eclectic Repertory*, October, 1812, he reported 2 cases in which he washed out the stomachs of children who had swallowed laudanum. These are the first instances in which lavage was practised for the purpose of removing poison from the stomach. At the time Dr. Physick was ignorant of the fact that lavage in such cases had been suggested by Alexander Monro, *secundus*, in his *Inaugural Thesis*, in 1797. Monro had, however, never used it. Physick published a note in a subsequent issue of the *Eclectic Repertory* stating that since his previous report he had read Munro's suggestion and hastened to give him credit for it.

In Physick's time it was customary to use silk or flax for ligating bloodvessels. These substances were not absorbable and consequently remained in wounds for weeks or months and, acting as foreign bodies, kept the wound from healing and caused prolonged suppuration. Physick wished to obviate these disadvantages and undertook a series of experiments with animal substances, as a result of which he used the latter entirely from 1816 onward.[1] The idea of the use of animal material for ligatures had been suggested previously by others, but Physick was ignorant of this when he conducted his experiments, and the latter were entirely original in idea and method with him.

In 1820[2] Physick published a method for the removal of scirrhous tonsils and hemorrhoids by means of a double cannula and soft wire. A few years later[3] he described a guillotine for the excision of the

[1] Eclectic Repository, 1816, vol. vi.
[2] Philadelphia Jour of the Med. and Phys. Sci., 1820, vol. i.
[3] Am. Jour. Med. Sci., 1828, vol. i.

uvula, of which he had devised a modification for the excision of the tonsils, and subsequently[1] he described a vulsellum forceps to grasp the tonsils in performing the operation. These instruments constitute the original models on which all the modern instruments for this purpose were designed. In former days skill in operations upon the eyes was frequently associated with similar ability in operating for stone in the bladder. Cheselden, the great English surgeon of the eighteenth century, possessed great fame in both these branches, and the same may be said of Physick. I will conclude my account of his achievements by the brief mention of 2 cases illustrating this point. In October, 1831, when Physick was sixty-three years old, he performed the operation of lithotomy on Chief Justice John Marshall, aged seventy-five, removing no less than 1000 calculi from the bladder of his distinguished patient. John Marshall died four years later of some hepatic complaint. At the autopsy his bladder was found absolutely free from calculi or any form of disease. The last operation performed by Dr. Physick was for cataract on a foreigner upon whom Physick had operated a year before. The patient had waited in Philadelphia a whole year for the second operation and Physick, though in wretched health and quite feeble, performed the operation rather than disappoint him. Randolph says: "The date on which he performed this operation was the 13th of August, 1837. I was present on the occasion, and watched him with the most intense anxiety. He was quite collected and firm, and his hand was steady, notwithstanding at the time he was laboring under great mental and physical suffering." Physick suffered from hydrothorax, which was so bad that he could not lie down to sleep. He developed a general anasarca and died after much suffering on December 15, 1837.

Apropos of the death of Physick one may recall a story told by Randolph: "On the evening of the day Dr. Rush was buried, Dr. Physick's doorbell was rung and he answered the summons himself. In the doorway stood a burly Negro, who demanded, 'Do you want Dr. Rush?' 'What do you mean,' said Physick; 'Dr. Rush is dead!' 'I will have him at the College for you at nine o'clock tomorrow morning for twenty dollars,'" said the Negro. Of course, the offer was not accepted, but Physick was so shocked that he took precautions that after his death his grave should be guarded against the possibility of his resurrection.

[1] Am. Jour. Med. Sci., 1829, vol ii.

The last of the Edinburgh graduates who were on the faculty of the University of Pennsylvania of whom I shall speak was the distinguished Caspar Wistar (1761–1818). Carson points out that as he had begun his medical studies in 1777 in the medical school of the College of Philadelphia and he graduated in 1782 from the University of the State of Pennsylvania, it was very natural that he should have been most active in the steps leading to the union of the two to form the University of Pennsylvania. Wistar began his medical studies with John Redman and the famous surgeon John Jones. After graduating from the medical school of the University of the State of Pennsylvania he went abroad and got his M.D. from Edinburgh in 1786.[1] While in Edinburgh he was elected president of the Edinburgh Medical Society, a great compliment to an American, particularly so soon after the Revolution, but Wistar's whole career indicates what a very delightful and popular man he must have been. In 1789 he became trustee of the College of Philadelphia and soon after professor of chemistry. In 1792 he was elected Shippen's adjunct professor in the chair of anatomy. Wistar was an able teacher. He had a great number of specimens and models prepared for the use of his students, many of the models having been prepared by Rush, the most skilful woodcarver of his day. At his death he bequeathed his large collection of preparations and models to the University, where they formed the nucleus of the great Wistar Museum, which is such a notable feature in the Univeristy's activities at the present day. Wistar made some very notable contributions to the correct knowledge of the anatomy of the sphenoid and ethmoid bones. His *System of Anatomy*, published in 1811, was the first American book of its kind and was held in just esteem for many years. Wistar was also an excellent botanist and geologist. The beautiful wisteria plant was named after him by the Abbé Correa da Serra. In 1815 he succeeded Thomas Jefferson as president of the American Philosophical Society. During his life Wistar used to keep a sort of open house on Sunday evenings for his scientific friends and any scientific strangers who might be visiting Philadelphia. Upon his death some of his friends in the American Philosophical Society decided to continue holding somewhat similar social gatherings on Saturday evenings, and thus arose the famous Wistar Parties, which form such a feature in Philadel-

[1] Thesis, *De Animo Demisso*.

phia's social life to the present day, a unique monument to the hospitable nature of a great man.

And now it is appropriate that I should close this Oration with a reference to the relationship that these men who had received such excellent training and shown such splendid results held to this our College of Physicians.

I shall begin with another quotation from Dr. Mitchell's beautiful address at its Centennial: "On the 2d of January, 1787, in the evening, in a little house used by the University and known as Surgeon's Hall, on Fifth Street, south of Library, assembled a portion of the notable group of men who then constituted this College. By the dim light of candles, for which I have found the modest bill, clad after the fashion of the day, some in Quaker dress and some in knee breeches, silk stockings and low shoes with buckles, most of them carrying, I fancy, the gold-headed cane and the meditative snuff-box, some with queues or powdered wigs, a fading fashion, were John Jones, William Shippen, Jr., Adam Kuhn, Benjamin Rush, Thomas Parke, Gerardus Clarkson, Samuel Duffield, James Hutchinson, William W. Smith, Andrew Ross, William Clarkson, James Hall, William Currie." The full roll of Fellows and Junior Fellows in January, 1787, adds the names of John Redman, John Morgan, George Glentworth, Abraham Chovet, Benjamin Say, Samuel Powell Griffitts, Benjamin Duffield, John Morris, John Carson, John Foulke, Robert Harris."

It seems to me I feel the thrill of pleasure that inspired Dr. Mitchell when he read this roll of twenty-four distinguished names.

Of the eleven Senior Fellows four were graduates in medicine from the University of Edinburgh, namely, William Shippen, Jr., John Morgan, Adam Kuhn and Benjamin Rush.

The following three Senior Fellows had studied at Edinburgh but for various reasons had not got their degrees from that University: John Redman, the first President of the College, graduated M.D. at Leyden in 1748; John Jones received his M.D. at Rheims in 1757; Thomas Parke, studied in both London and Edinburgh, but was content throughout his professional life with the degree of Bachelor of Medicine, which he received from the College of Philadelphia in 1770.[1] Thus seven of the eleven were Edinburgh men.

The first Secretary of the College, James Hutchinson, had gone

[1] Gerardus Clarkson studied abroad "for a prolonged period," but I cannot ascertain where.

to England and begun his studies under the auspices of Dr. John Fothergill, but had been compelled to return to America because of the outbreak of hostilities between the Colonies and Great Britain. On his return voyage he had been entrusted by Benjamin Franklin with important papers for Congress. When off the American coast his ship was pursued by a British warship. Hutchinson threw himself into a small boat and got ashore under heavy fire. The ship was captured and he lost all his personal belongings but he had saved his despatches.

Fellows of the College and Guests: These were the men whose tradition we have inherited, and no one better exemplified their importance and true worth than our great Fellow, Weir Mitchell. His many-sided career presents many points of resemblance to that of Benjamin Rush. Both were not only great physicians, but also were especially distinguished as students and teachers of neurology. Both were interested in much outside of their strictly professional activities, were animated by ardent zeal for the welfare of their fellowmen, and proud of their profession and jealous of its dignity. How much this College owes to Weir Mitchell can never be adequately expressed, but the greatest debt of all is probably that we owe to his zeal for the preservation of the highest professional ideals which he received from his predecessors and maintained throughout his life.

DIVERTICULOSIS OF THE ALIMENTARY AND URINARY TRACTS*

THE MÜTTER LECTURE

BY EDWIN BEER, M.D.
NEW YORK CITY

As one surveys the literature of medicine, one cannot fail to be impressed with the need of the closest coöperation between its many sister sciences. The study of pathological anatomy has led to many clinical advances, and, on the other hand, the observations in the clinic have stimulated pathological and chemical researches. If one is optimistically inclined, one cannot fail to conclude that many of the pathological conditions that were not observed either in the clinic or in the autopsy room, really existed with about the same frequency as we observe them today, but, owing to human oversight or error, they were not recognized. On the other hand, if one is more inclined to pessimism, one might conclude that the human race is degenerating and gradually succumbing to more and to newer pathological manifestations. Perhaps there is some truth in both viewpoints. Not so long ago ulcer of the duodenum was considered an unusual entity,[1] whereas gastric ulcer was considered much more common. Was this due to oversight and to human error in observation? Again, bladder tumors were considered a rare condition at the commencement of this century, and now, since the clinician knows how to recognize them, their frequency is quite appalling. Perhaps in no group of diseases is this apparent increase more striking than in the subject of tonight's presentation. Prior to the commencement of this century, diverticula of the hollow viscera, exclusive of diverticula such as Zenker's and Meckel's, diverticula of the colon and of the urinary bladder were practically unknown in the clinic and only rarely reported by the pathologist, even though the diverticulum associated with the name of Meckel had been described in 1671 by Morgagni. Such diverticula now-

* Read December 2, 1931.

adays are so well known to the average clinician, and naturally to the pathologist, that it is with some hesitation that a review of this subject is presented tonight. It is difficult to believe that these conditions are new entities—that they have increased in numbers so rapidly that now thousands of cases are on record. When I collected all published cases of diverticulitis[2] of the sigmoid, etc., in 1904, there were only 18 clinically observed cases recorded. About ten years later, when I made a similar review[3] of the somewhat analogous conditions in the urinary bladder, there were only 19 cases recorded in the clinical literature. In the intervening period the literature has been almost flooded with additional cases of both intestinal and bladder diverticula, and some clinics have reported series varying from over 100 cases to over 1000 cases of these interesting conditions, cases which have been carefully observed and often treated with great success. I cannot but feel that the optimistic attitude referred to above, even though it carries with it a certain amount of criticism of our predecessors, is justified, and that the discrepancy between the incidence of the disease today and thirty years ago is attributable to the human-error factor. It is difficult to explain this discrepancy on the basis of increased incidence of these diseases, though the prolongation of life and the great increase in the population may have influenced to some extent the marked increment. Newer methods of investigation, new instruments of precision, have made the recognition of these pathological conditions possible and the roentgen-ray, above all, has thrown a bright light into this whole field and established with great accuracy the frequency of these curious pathological entities.

Diverticula have been observed in all parts of the alimentary tract from the pharynx to the rectum, as well as in the urinary tract from the ureteropelvic junction to the penile urethra. In both these systems many interesting and instructive analogies are observed and by a study of these diverticula in both systems a somewhat clearer insight as to their causation is obtained. Considerable difference of opinion has been voiced as to the factors that lead to diverticula formation; some authors believing that all, or most, are acquired; others that most are congenital, and others again combine the two viewpoints. From a study of the anatomy of the diverticula and the recognition of all the walls of the organ from which it is derived, *i. e.*, mucosa, submucosa and muscularis, one can fairly conclude that such a diverticulum is of the congenital

type, corresponding as it does in structure with Meckel's diverticulum and those found in fetuses or in early life. Occasionally adjacent disease process may draw on one of the hollow viscera and produce by traction a diverticulum having these same anatomical characteristics, and, though it is evidently acquired, its structure may closely simulate the congenital type. On the other hand, many diverticula, in fact most of those that I have seen, have no such regularly disposed layers as the definitely congenital; they are composed of mucosa, more or less submucosa, and here and there smooth muscle bundles. Are these to be considered congenital diverticula in which atrophic changes have taken place as a result of the pressure changes and subsequent stretching of these pouches, or are they all acquired? I believe many, especially in the urinary bladder, are to be interpreted in the former sense; and others, perhaps the great majority in both the alimentary and the urinary tracts, in the latter sense.

The analogy between the small diverticula that develop as multiple herniations of the mucosa between the hypertrophied and separated muscle bundles in the bladder and those that occur in the colon, especially in the sigmoid, is most striking. They both occur in organs that act as reservoirs, in which pressure changes are frequent, whether urine, feces or gas, produce these changes. They develop usually in adult life in patients who have obstructive conditions in the urethra or in patients who have some intestinal disturbance, whether flatulence or constipation, associated with a possible hypertonicity of the anal sphincter. In both systems they are hernial protrusions of the mucosa with little or no evidence of muscle tissue in their walls. Whether the vascular structures that perforate the walls make for weakness where herniation occurs has not been proven, even though such vessels are often adjacent to the diverticula. Perhaps they lead the way after the weakness in the connective tissue or muscular tissue allows of the inception of the process. Congestive cardiac disease and portal obstruction do not seem to predispose to their development, as would be expected if the weakness was primarily where the vessels perforated the involved hollow viscera. This type of diverticula formation has all the earmarks of an acquired process. The same process in the esophagus seems to produce the pulsion diverticula between the inferior constrictor muscle and the cricopharyngeus at the upper end of the esophagus. Here the trauma of hasty deglutition (bolting

of food) seems to force the weak point and gradually a pouch is acquired which presents the definite picture of a diverticulum.

Between these rather definitely congenital and acquired diverticula there is a large group of diverticula, particularly in the urinary bladder, which are difficult to classify. The same applies to those diverticula in the stomach, duodenum and small intestine, as well as in the ureter and urethra, in which the pouch formation is not due to giving away of the wall due to inflammatory disease, as in ulcers or adjacent to ulcers. In the urinary bladders we find with some frequency single and multiple diverticula, which are usually much larger than the mucosal herniations referred to above which bear such similarity to the sigmoid diverticula, and many of these show but scanty evidence of smooth muscle tissue in their walls. They are at times seen in early life (I have excised several such diverticula in young children), but may occur or be found at all ages and are frequently associated with obstruction at the neck of the bladder, either contractures or prostatic enlargement. In the children that have had this condition, no evidence of obstruction was detected by me. These diverticula may grow to large size; one recently recorded held 5 liters of urine.[4] It must be apparent that this group is totally different from the group previously described, which resembles so closely the diverticula of the colon, and the question is still under discussion as to whether they owe their origin to a congenital fault or not. In view of the fact that they arise so regularly close to the normal ureter opening (in some rare cases the ureter empties into the pouch quite a distance from the lumen of the bladder), one cannot but feel that there is a congenital "anlage" for their development, whether in the form of an extra ureter "bud" or otherwise. The increase in size would gradually develop as a result of the pressure changes in the bladder, and what may have been imperceptible at birth, or only a dimple, gradually grows in size, so that it can be recognized in childhood or adult life as a sizable diverticulum. A similar embryological explanation has been offered for most of the diverticula of the small intestine, but here owing to absence of adequate pressure changes the large pouches seen in the bladder do not develop.

All the above three types (the congenital, the acquired and the congenital, that have evolved acquired characteristics) are fairly considered true diverticula, as opposed to those false diverticula which result from traction from without, from disease in the organ

involved or from adjacent disease or trauma which perforate the organ that develops a diverticulum. Abscesses may extend into a hollow viscus and communicate with same, producing in this way an artificial pouch. Trauma, such as operative repairs of vesico-vaginal fistulæ, may artificially produce a similar pouch with all the signs, symptoms and possible complications that arise in any true diverticulum.

The most interesting diverticula, as well as those most frequently encountered in our clinical work, are: those occurring in the pharyngoesophageal area; those occurring in the intestine, especially in the colon and those occurring in the urinary bladder. In the stomach, small intestine (excluding Meckel's diverticulum), rectum, ureter and urethra the condition is apparently somewhat of a rarity, and this is probably not due to the human-error factor, as for a number of years the possibility of their occurrence has been in the mind of the clinician and adequate search has undoubtedly been instituted.

All diverticula, irrespective of the organ from which they arise, are prone to similar pathological changes in the course of their development. Their growth is usually slow, and only in the esophagus, duodenum and bladder do they grow to any size. Irrespective of their size, they are exposed to trauma and infection, and both these conditions combined may lead to serious complications. Meckel's diverticulum occupies a peculiar position in this whole group, as, owing to its varying anatomy and adhesions, at times it leads to intestinal obstruction, acting as a band, or even gets tied into most bizarre knots that lead to obstructive conditions and to gangrene. Another peculiarity of this congenital diverticulum is the finding in its mucosa of typical bleeding peptic ulcers which are caused by or derived from misplaced gastric mucous membrane. Other peculiarities of this diverticulum are its opening by a narrow or wide channel at the umbilicus. The other diverticula, except those rare bladder urachal pouches, never empty primarily at the navel nor are liable to produce the above intestinal complications. Foreign bodies are liable to get lodged in these pouches and may lead to perforation with localized or diffuse infection. Even without the introduction of foreign bodies, inflammation and ulceration are liable to develop and these in turn lead to more or less extensive destruction of the various walls of the diverticula and to peridiverticulitis and adhesions to adjacent tissues and organs, without

or with perforation into such organs. In the colon or the bladder, long stagnation may lead to inspissation of the contents of the diverticula or the development of calculi, and quite regularly one finds small hard fecal masses in the acquired diverticula of the colon, much less frequently stones in those developing in the urinary bladder. As a result of the chronic irritative process, occasionally neoplasms develop in these pouches and both benign and malignant growths, carcinomata and more rarely sarcomata have been encountered.

Undoubtedly the great majority of diverticula do not give clinical evidence of their existence and are recognized only in careful routine examinations. When inflammatory changes set in, they often begin to give symptoms and cause trouble. Esophageal diverticula may give symptoms from pressure, and Meckel's and duodenal diverticula may also produce symptoms by mechanical interference with the intestinal lumen or by pressure on adjacent ducts. All others become vocal when infection develops, which may lead to local symptoms or at times to reflex symptoms suggestive of gastroduodenal disease.

Diverticula of the Stomach. According to recent study[5] the earliest mention of such a diverticulum was by Helmont in 1804. Only a few hundred are recorded in the literature and most of them have been recognized during routine roentgenological studies of the stomach and intestines. At operation they have been rarely encountered and at autopsy a few have been recognized. Most of the cases have been found in adults. Apparently the true diverticula are more common at the cardia, where they are composed of all the coats of the stomach and are possibly congenital. In the distal part of the stomach, diverticula resulting from ulceration are found, presenting the picture of false diverticula. The frequency of the "niche" formation in ulcer bases is well known and may be the starting point of some of these acquired pouches. Symptomatically these diverticula, unless associated with other pathological changes, give rise to little disturbance. If associated with these changes, which may lead to perforation or abscess formation, surgical therapy may be necessary.

Diverticula of the Duodenum, Jejunum and Ileum. These diverticula are more common than those in the stomach, and those in the duodenum have been recognized with increasing frequency since radiographic investigations are routine. J. R. Case diagnosed

85 cases in this way. In a recent German publication, another series of 50 duodenal diverticula was recognized in this way. Diverticula in the rest of the small intestine are rarely visualized in this manner. In some diverticula an air bubble has been seen above the opaque barium.[6] False diverticula also occur in the duodenum secondary to ulceration, and at times between the ulcer and pyloric ring. The anatomy of these latter pouches[7] has not been described as far as I know. Those occurring in ulcer bases or adjacent to these are similar to the same process in the bases of gastric ulcers. According to recent publications, about 50 cases of diverticula of jejunum and ileum (excluding Meckel's diverticulum) have been noted and the roentgenographic diagnosis was reported as made in only 3 cases.[8] This is evidently inaccurate, as a perusal of American literature shows.[9] Whereas in the duodenum these diverticula are usually single, in the lower part of the small intestine they are usually multiple and in 1 recent case over 300 such pouches were recorded. There is considerable difficulty in explaining those diverticula that do not develop from ulcer bases. Some authors think misplaced pancreatic tissue which is occasionally found in their walls suggests that developmental anomalies underlie the process, especially below the duodenojejunal junction. Atavistic influences would thus underlie the development of these curious structures. Others believe there is a primary connective tissue weakness which predisposes to the mucosal herniation. In the various parts of the duodenum, diverticula develop more frequently near the entrance of the ducts than in parts one and three and often grow into the head of the pancreas. In the jejunum and ileum they seem to develop almost regularly between the leaves of the mesentery, differing in this way from Meckel's diverticulum, though some have been noted on the convexity of the bowel and pancreatic tissue, or neoplasms have been found in their walls.[10]

Symptomatically these diverticula are liable to be quiescent unless inflammatory changes take place in them or a foreign body is caught in them and leads to perforation with abscess formation or to perforation into the peritoneal cavity. In duodenal diverticula, reflex gastric symptoms somewhat suggestive of gastroduodenal ulceration are not uncommon. Occasionally obstructive symptoms, with or without jaundice, develop. In view of these varied symptoms a fair number of duodenal diverticula have been operated upon, either by resection or by infolding. Whether any of these

operated cases have subsequently recurred the literature does not make clear, though early control pictures have given no indication of recurrences. In the ileum, similar resections of the pouches have been made, and in a few cases of multiple extensive diverticulosis of jejunum or ileum segments of bowel have been resected.

Meckel's Diverticulum. This congenital pouch occurs usually in the last part of the ileum, within one yard of the ileocecal valve. Though usually small, about the size of a plum, it may be 30 cm. in length. Many of its peculiarities have been referred to in the beginning of this paper. Its frequency is much greater than those in other parts of the small intestine, being found, according to the statistics of the British Anatomical Society, in 2 per cent of autopsies. Considering this incidence, it is surprising how rarely it gives rise to symptoms and how infrequently surgical reports of its removal are published. For many years, in every so-called interval appendicectomy that I performed, I examined the lower ileum for such a diverticulum and never encountered one. On the other hand, in a similar series of 507 cases, R. M. Harbin[11] has found Meckel's diverticulum in 7 cases.

The many bizarre pathological pictures produced by this vestigial organ are fairly well known, even though no one has encountered all of them in his own experience. Derived as it is from the omphalomesenteric duct, it may remain open all the way to the navel and produce a mucous membrane sinus either connecting with the ileum or ending as a blind pouch. Cysts or tumors of various kinds may develop in the peripheral or proximal end, and at times the prolapsing mucosa may present gastric mucosa. If the peripheral end is well closed, it occasionally is represented by a fibrous band which may lead to intestinal obstruction, as loops of gut become engaged under the band thus formed. Even if no such band exists, similar obstructions are caused by the diverticulum itself, especially if it is adherent or twists itself into knots of most surprising pattern. These peculiar gyrations may lead to strangulation and gangrene of the diverticulum itself; in other cases to strangulation and gangrene of other loops. At times it invaginates into the ileum and leads to extensive intussusceptions. Repeatedly it has been encountered in hernial sacs. Perforation by foreign bodies which get lodged in the diverticulum is no great rarity and hemorrhage from ulceration, usually due to misplaced gastric mucosa, is being more frequently recognized and reported. Perforation into the bladder is a rarity.

Fortunately the neck of these diverticula is usually wide and their muscular wall is able to keep them fairly empty, so that inflammatory changes are uncommon. If their frequency at postmortem is as great as reported by the English Commission and others,[12] it is surprising how rarely one is called upon to treat their intrinsic disease or conditions secondary to their presence.

The diagnosis of Meckel's diverticulum is at best a good guess, as no pathognomonic features are known. As far as I know, radiographic studies have given no conclusive data, which is surprising in view of their frequency.

At operation, ablation or invagination (in small diverticula) have been carried out. In the more complicated cases with other secondary pathology, further surgical measures must be taken as indicated by the findings.

Diverticula of the Appendix. These diverticula[13] are rather infrequent and almost always of the acquired type, resembling the mucosal herniations found in other parts of the colon. They are probably secondary to previous attacks of inflammation and naturally predispose to further trouble in this organ. Some of the pouches may lead to retention cysts and simulate mucoceles of the appendix, and when they rupture into the free cavity may give rise to the rare entity known as pseudomyxoma of the peritoneum. They are accidental findings at operation or at autopsy and practically impossible to recognize by radiography.

Recently, in connection with a study of cecal diverticula, a review of these structures has been made and emphasis has been placed on the possibility of acquired diverticula developing in the base of an excised appendix.[14] Apparently the purse-string closure of the cecum over the ligated appendix stump predisposes to this type of pouch formation, sometimes following suppuration about the ligated stump and other times even without such suppuration. By crushing the stump and ligating with plain catgut, which is rapidly absorbed, the purse-string-suture method will probably but rarely lead to such artificial diverticula formation.

Diverticula of the Rectum. In this portion of the intestinal tract, possibly because of its excellent support and fixation, diverticula seem to be a great rarity. The literature contains very few reports of this condition, which may be in part due to the fact that a careful postmortem examination of the rectum is not regularly made. Inflammatory changes and malignancy have been reported

in connection with the few cases recorded, and the diagnosis is practically impossible except after careful dissection of the whole excised rectum.

Diverticula of the Ureter. In this structure pouch formation is very rare, apparently only 12 have been recorded in the literature.[15] This may be, in part, due to the difficulty in recognizing these structures by our older retrograde methods. It is just possible that continued use of one of the 7 or 8 intravenous or oral urographic media will readily demonstrate more of these pouches. Whether these diverticula are congenital or acquired is not known. Obstructive conditions and stones probably lead to some of them, and these seem to be definitely acquired, though in others the structure is identical with that of the ureter, which suggests an embryonic fault, perhaps an undeveloped branch of a bifid ureter. Apparently they occur anywhere from the ureteropelvic junction to the insertion in the bladder, and if they are responsible for symptoms and protracted infection, operative excision or more radical procedures will be necessary.

Diverticula of the Urethra. In the urethra pouch formation is much more frequent than in the ureter. Almost all cases are reported in males, though recently some diverticula have been discovered in the female urethra.[16] In 1908[17] some 69 cases were collected from the literature. They develop in the floor of the urethra, and in the great majority of cases are acquired, though some congenital diverticula have been described in the region of the fossa navicularis, probably due to poor union of the penile urethra with that of the glans. Acquired pouches are due to a variety of causes and are found in any part of the floor of the urethra. They may be the result of retention cysts which rupture into the urethra; they may be due to periurethral suppuration of varying etiology, and they may result from injuries, accidental or operative. As they may lead to retention of infectious material, a urethritis may become intractable. Calculi have been encountered tucked away in these pouches. Some rare diverticula have been recorded as large as a fist, though usually they are no larger than a lima bean. Pouches of any size may cause obstruction and retention of urine. The diagnosis is by no means simple, even though the penile diverticula may be palpated along the ventral surface of the organ. Endoscopy and radiography of the urethra, after injection of opaque substances, will establish the diagnosis and site of the pouch.

Surgical removal may become necessary and the difficulty of the operation will vary with the location of the pouch.

All the above interesting diverticular formations are rarities in comparison with the three groups that follow. These latter we all encounter with considerable regularity, while the groups described are "rara aves" in most clinics.

Diverticula of the Esophagus. These pouches, like most diverticula, occur in adults almost exclusively and more frequently in males. They usually develop in a weak area between the lowest circular muscle fibers of the inferior pharyngeal constrictor and the adjacent oblique fibers on the posterior aspect of the tube. These upper esophageal pouches are therefore really pharyngeal diverticula, though long-established custom has called them esophageal or Zenker diverticula. An occasional case of diverticulum formation has been described where the pouch was just cephalad to the diaphragm. Between these two areas, near the level of the left bronchus, traction pouches develop as a result of adjacent lymphatic or mediastinal disease which pulls upon the esophagus. The former type has long been recognized as a "pulsion" diverticulum and the latter as a "traction" diverticulum. The study of these pouches has to some extent affected the interpretation and nomenclature of diverticulosis in other parts.

Those diverticula that develop in the upper part of the tube, in the hypopharynx, usually present on the left side of the neck and may grow into the thoracic cavity. They show in their walls mucosa and submucosa covered by a more or less thick fibrous adventitia. If long-standing inflammation has been present, they may become quite adherent to the adjacent structures, but usually are readily freed, being loosely attached. The mucosal side may show varying degrees of inflammation, secondary thickening, ulceration or neoplastic changes. Rarely, if not intrumented, does perforation take place. These pouches produce symptoms by pressure on the adjacent esophagus and by reflex spasm of its superior sphincter (the cricopharyngeus muscle), as well as by the fermentation and putrefaction of the food that stagnates in them. Obstruction to swallowing and slowly developing inanition are the outstanding symptoms. Dysphagia and the discharge of ingested food by pressure on the more or less definite tumefaction on the side of the neck are characteristic of the disease. Instrumental diagnosis

with sounds or bougies is rarely necessary, since esophagoscopy and radiography are available. Roentgenological observation and films after barium meals have displaced almost completely all the earlier diagnostic procedures, though esophagoscopy with proper instruments is more valuable in determining the extent and character of the changes in the mucosa lining the diverticulum. As many of these cases come for treatment in varying degrees of malnutrition, they may have to be built up before any radical therapy can be instituted. Medical treatment and even instrumental stretching of the more or less spastic and deformed entrance of the esophagus adjacent to the neck of the diverticulum has rarely had any lasting influence. Operative excision alone seems to promise real definitive cure. Preoperative feedings through a small-calibred tube introduced through the nostril into the stomach, using if necessary the string technic to guide the tube past the diverticulum, should be tried before resorting to gastrostomy in cases with severe or extreme inanition caused by long-standing obstruction. The endoscopic route for removal, by invagination or for establishing a lateral communication between diverticulum and esophagus, seems too dangerous to be recommended. Even if periesophagitis, cellulitis and mediastinitis could be avoided, recurrence or palliation is the best that such methods could offer. In larger pouches excision in one or two steps seems the only rational procedure. Though one group of surgeons favors the one method and another favors the other, it is difficult to be convinced that the mortality of the one-step[18] is greater than that of the two-step procedure and that fistulæ are not much more frequent after the two-step operation than after the one-step. The various refinements of these operations need not be discussed at present. It must be apparent that, as far as recurrence is concerned, a clean-cut one-step excision without interference with the esophageal lumen either by traction or by suture should guard the patient against future trouble. The dangers of cellulitis and pulmonary complications must be reckoned with in all surgical procedures on these more or less infected pouches.

Those rare diverticula near the lower end of the esophagus are rarely encountered and very rarely grow to any size. Symptoms also are unusual unless cardiospasm is present. In 1 recent case of this type, care in diet and antispasmodics gave relief. In the very unusual larger pouches various operations—excising (W. Meyer and Sauerbruch) or anastomosing the pouch with the stomach—

have been suggested, but as far as I could review the literature, only 2 excisions and 1 anastomosis (Henschen) have been performed successfully in diverticula of the lower end above the diaphragm.

In the mid region the small traction diverticula also rarely cause symptoms, even though autopsy examinations show them to be the most frequent type in the esophagus. They usually are composed of the walls of this organ and lie horizontally or run obliquely upward toward the lymph pocket that has distorted the esophagus. Occasionally, if they are so placed that food can enter them, they may enlarge under the influence of pulsion and a so-called pulsion traction pouch develops. The diagnosis of these diverticula and those at the lower end of the esophagus are made by radioscopy. In the absence of symptoms no therapy is called for except possibly dietetic management.

Diverticula of the Colon. The group of colonic diverticula constitute one of the most important manifestations of this pathological entity. It took ages before inflammatory processes in the right iliac fossa were properly explained and connected with inflammation of the appendix and it took even longer before similar processes in the left iliac fossa were recognized as due to diverticulitis of the lower colon. These diverticula are very frequent, occurring in 5 to 10 per cent of all adults, and may occur in any part of the colon from the cecum to the rectum. They are almost invariably multiple and frequently very numerous. Only a small percentage of diverticula seem to produce symptoms.[19] At times diverticula in the small intestines are found associated with those in the colon. In the sigmoid and descending colon they occur most frequently and it is here that inflammatory changes are most commonly found. Although this is a disease of adult life, a few cases of colonic diverticula have been found in the young. Males are more frequently affected and most patients complain of flatulence or of constipation. Apparently these pouches are herniations of the mucosa through weakened areas of the intestinal wall, and develop either along the longitudinal bands, into the appendices epiploicæ or into the mesenteric attachment as the result of pressure changes within the colonic reservoir above a torpid rectum or a spastic, tight sphincter. They rarely are larger than cherries and often much smaller. Intestinal contents collect in these pockets and, owing to the narrow necks of the diverticula as well as to the almost complete lack of musculature, stagnation and inspissation result. This favors local irritation,

inflammation and ulceration, with fibrous tissue reaction. If these processes are too rapid for Nature's reaction the ulceration may lead to perforation into the peritoneal cavity or retroperitoneally, which fortunately is a rare occurrence. Usually the process is slow, diverticulitis leads to peridiverticulitis with local suppuration or interstitial inflammation in the colonic wall and mesentery. Some thirty years ago I happened to see 2 cases of abscess in and about the sigmoid, associated with tumefaction and incomplete obstruction, which had simulated clinically carcinoma of the lower bowel. On drainage of the perisigmoid abscess the condition slowly cleared up. These unusual cases seemed quite inexplicable at the time, but shortly thereafter, while engaged in autopsy work in Prague, I found the probable explanation for the above cases in the inflammatory reaction that I saw in and about the sigmoid in infected diverticula. It was as a result of these observations that I collected all the clinical and pathological data already recorded and published them in 1904. In some cases the inflammatory reaction leads to such extensive tumefaction with obstruction of the lumen that the clinical picture suggests neoplasm. At times the inflamed sigmoid becomes adherent to adjacent viscera, most commonly the bladder, and a fistula develops between these organs. Rarely malignancy arises in these diverticula, and then both the clinical and the pathological diagnosis may be most difficult.

The diagnosis of diverticula of the colon may be suspected when the above pathological changes are in evidence; but without the colonic roentgen-ray and the barium meal the diagnosis is at best only a good guess. Sigmoidoscopy is in general useless, except in so far as it may detect a diseased mucosa which is rarely present in diverticulitis, as this produces interstitial mural changes rather than changes in the mucosa. Direct inspection of the bowel may discover a neoplasm, which may account for the symptoms and thus establish the diagnosis. In those rare cases of sigmoidovesical fistula, cystoscopy with or without excision of mucosa adjacent to the opening in the bladder may be helpful in excluding tuberculosis and carcinoma, which are the other more common diseases that lead to the condition. The radiographic picture is particularly clear after the bowel is evacuated and the residue that remains in the pouches is visualized as opaque circular shadows along the course of the colon. Whether the prediverticular stage can be recognized radiographically is open to doubt. Its characteristic appearance

is said to manifest itself by loss of normal segmentation, more rigid outline and small convexities in the outline.[20]

For a complete radiological study, both the meal and the enema seem indicated. Following the barium meal the above globular bodies may be detected many days after the rest of the colon is empty. With the barium enema similar pictures may be seen after evacuation, and during the filling one usually sees the irregular stockade-like or serrated outline of the involved areas.[21]

As said above, only a small percentage of diverticula become inflamed and require either medical or surgical treatment. The former consists in simple diet with lubrication and irrigation of the intestine, so as to put the parts at rest and prevent further irritation. The latter consists in meeting the conditions presented at operation, such as drainage of local abscesses or treatment of a free perforation by closure and drainage; cecostomy to put the inflamed sigmoid or colon at rest; in suitable cases a Vorlagerung of the involved gut with secondary excision, or separation of bladder and sigmoid in fistulous cases with closure of both perforations and interposition of omentum to prevent reanastomosis of the two involved and much indurated viscera.

Diverticula of the Urinary Bladder. Next to the group just described, diverticula of the urinary bladder are the most frequent seen in the modern clinic. They occur in all ages, though definitely much more common in adults and advanced life. They are very rare in the female, probably because of the rarity of obstructive conditions at the bladder outlet. The small multiple hernial protrusions of the mucosa, seen so often in prostatic obstruction, resemble closely the sigmoid and colonic cases just described. They are found all over the bladder convexity and develop between separated hypertrophied bands of musculature. In the cystogram they may produce a serrated outline very similar to that seen in the sigmoid, but here the serration is definitely due to the filled diverticula and are much more blunted or rounded. Infection with perforation into the free peritoneal cavity is very rare, but perivesical inflammation is probably much more frequent than is realized in the clinic. Stones corresponding in size to these small pouches may develop in them, and in 1 case, by pressure here and there against the bladder wall with a blunt instrument within the viscus, I have squeezed out many dozen small stones without any difficulty, each stone jumping out into the bladder like a pea out of its pod.

All these diverticula seem to be acquired and secondary to obstruction to the outflow of urine.

In contrast to these small diverticula, which rarely are larger than a walnut and usually much smaller, are those interesting large diverticula which are at times single, but often multiple, that seem to be congenital in origin. They occur in young children as well as in adult life. They may be enormous, holding 5 liters of urine. One that I removed was as large as the distended bladder and held 26 ounces. They probably owe their origin to some congenital anlage or malformation and usually grow in size as the individual ages. Obstruction to the outflow of urine from the bladder is not always present; but when it is, growth is probably accelerated and large diverticula present. These diverticula always arise near the ureteral orifices and, as they grow, they occasionally draw the ureteral mound and its orifice into the sac. Insertion of the ureter into the diverticulum at some distance from the bladder wall is much less common, only 1 such case having been encountered by me. Inflammation in these diverticula is liable to develop and peridiverticulitis may involve the adjacent lowermost ureter so that it seems to be part and parcel of the wall of the sac. Stones, at times collar-button stones, part in the diverticulum and part in the bladder, are liable to develop. In other cases, faceted groups or single stones are found in these pouches. Leukoplakia and neoplasms arising from the diseased mucosa of these diverticula are by no means rarities. Infection in these structures is practically uncontrollable, as they usually have little or no musculature, if they are of any size, and therefore they cannot empty themselves. Their presence disturbs the bladder mechanism as the detrusor forces the urine into the diverticular cavity at each micturition. This disturbance is usually followed by hypertrophy of the bladder musculature and occasionally by giving way of the ureter orifices and dilatation of the upper tract on one or both sides. In 1 case of large diverticulum which was filled for roentgen study with argyrol, I found the argyrol still in the diverticulum thirty-five days later, though the patient had bladder irrigations almost daily. The presence of such an infected diverticulum may prevent the smooth closure of a suprapubic bladder wound.

Unless infection is present the presence of these pouches may never be suspected. Rarely they are recognized by palpation either in the suprapubic area where a spherical mass may be pal-

pated after micturition. Several cases in children were detected in this way. By rectum one can also at times feel such a filled diverticulum. The only reliable diagnostic aids, however, are the cystoscopic examination and the roentgenological demonstration with opaque fluids, such as sodium iodide, sodium bromide and the like, the pictures being taken anteroposteriorly and obliquely. Frequently, after emptying the bladder, an aërogram of this organ will show the diverticula still fairly well filled with the opaque solution, while the bladder is distended with contrasting air. With the cystoscope, one regularly sees the dark orifice of the diverticulum, though in a few cases, what looks like a sphincter closure may temporarily prevent visualization of the orifice.

In the first group of bladder diverticula so frequently seen in prostatic cases, rarely is surgical treatment required unless they harbor stones or are unusually large. Persistent moderate pyuria following adequate relief of obstruction is frequently due to persistent infection in these pockets. On the other hand, the second group demands surgical excision with adequate care of the ureter if it is involved in the process. Numerous methods of approach for the operative removal have been suggested, both from within the bladder and from without. My impression is that surgical exposure from without, after adequate mobilization of the bladder with visualization of the ureter or ureters, with careful external suturing of the bladder wall is the best approach and the surest method of preventing a recurrence. Almost twenty years ago, in doing the operation in this way, I became acquainted with the procedure of complete mobilization of the bladder, stripping it out of its peritoneal covering and its perivesical tissues, which technic I have used since then in my operative work in bladder resections for infiltrating carcinomata.

It must be evident that any method that fails to expose the orifice of the diverticulum from outside the bladder will not permit of accurate closure of the neck and predisposes to recurrence, a sequence that has been reported every once in a while in the literature. If the ureter opens near the orifice it can be turned into the bladder with a small flap to which it is attached. In those rare cases when it opens into the diverticulum at some distance from the bladder wall, it can be covered with a triangular flap of diverticular wall and thus introduced into the viscus before the orifice in the bladder wall is closed from without. This triangu-

lar flap has its base at the bladder, and as the ureter orifice is introduced into the viscus the edges of the flap are sewed over the exposed ureter, which then projects well into the bladder. The results of these operations are highly satisfactory and the mortality is low.

From this review it is apparent:

1. That diverticula of the alimentary and urinary tracts are much more common than was thought only a few years ago.

2. That diverticula may produce clinical symptoms and with our latest methods of investigation they can almost regularly be recognized.

3. That relief by surgical removal or analogous procedures is usually obtainable, without undue risk.

4. That diverticula in general are acquired, though in many cases they may be congenital or due to a congenital "anlage" and develop only when the factors that make for an acquired diverticulum are present.

(To illustrate the various types of diverticula over fifty lantern slides were shown.)

BIBLIOGRAPHY

1. Osler: Practice of Medicine, 1897, reports only 9 cases that came under his observation.
2. Am. Jour. Med. Sci., July, 1904 (Collected Papers, P. Hoeber, 1931, p. 3.)
3. Ann. Surg., November, 1913. (Collected Papers, P. Hoeber, 1931, p. 479.)
4. Kroh: Cent. fur. Chirurgie, 1931, p. 1731.
5. Palmer, D. W.: Mayo Clinic, 1930, vol. xi.
6. Ann. Surg., vol. lxxiv, p. 101; J. R. Case, Jour. Am. Med. Assn., vol. lxxv, p. 1463.
7. Crohn, B.: Affections of the Stomach.
8. Gisbertz: Zentralblatt fur Chirurgie, 1931, p. 892.
9. Stetten: Ann. Surg., vol. lxxiii, p. 241; Ann. Surg., vol. lxxiv, p. 101; J. R. Case, Jour. Am. Med. Assn., vol. lxxv, p. 1463.
10. Hansemann: Virchow's Arch., 1896, vol. cxliv.
11. Harbin, R. M.: Surg., Gynec. and Obst., 1930, li, p. 863.
12. Wisely, A. N.: Jour. Am. Med. Assn., vol. xcvi, p. 1949. Ewing, 2.7 per cent; Telling, 39 in 13,068 autopsies; Balfour, 15 in 10,600 laparotomies.
13. Sauer, K.: Am. Jour. Surg., 1930, p. 564. In 12,889 cases, 35 instances.
14. Greenberger and Hiller: Surg., Gynec. and Obst., 1928.
15. Chwalla, R.: Zeitsch. f. Urologie, 1930, vol. xxiv. H. Bugbee, Jour. Urol., 1931, vol. xxvi; J. H. Neff, South. Med. Jour., 1921, vol. xiv.
16. Furniss: Jour. Am. Med. Assn., 1931, xcvii, 1350.
17. Ritter and Leidel, G.: Arch. f. Klin. Chir., 1930, vol. clxiv; E. Langer, Zeit. f. Urol., 1930, xxiv, 519.
18. Lotheisen, G.: Ergebnisse d. Chir. and Orthopedic, 1930, vol. xxiii, summarizes 60 one-step operations between 1924 and 1928 with only one fatality.
19. According to W. J. Mayo, 10 to 12 per cent require surgery, Brit. Med. Jour., September 28, 1929.
20. Spriggs, E. I.: Brit. Med. Jour., September 28, 1929.
21. Case, J. R.: Am. Jour. Surg., New Series 4, No. 6, p. 573.

MEDICAL SCIENCE IN THE SERVICE OF THE STATE: SOME PROFESSIONAL EXPERIENCES*

THE MARY SCOTT NEWBOLD LECTURE XXVI

BY GEORGE BURGESS MAGRATH, A.M., M.D.

INSTRUCTOR IN LEGAL MEDICINE, HARVARD UNIVERSITY; MEDICAL EXAMINER FOR SUFFOLK COUNTY, MASSACHUSETTS; CONSULTING MEDICO-LEGAL PATHOLOGIST

Dr. Magrath gave a most interesting lecture on his personal experiences. It was delivered informally, without the aid of manuscript or notes, and was explanatory of the many lantern-slide illustrations which were shown on the screen. Each of the episodes described had all of the lively interest of an exciting detective story.—EDITOR.

* Delivered March 4, 1931.

APPENDIX

INTRODUCTORY NOTE TO THE SECTIONS

Almost all of the papers read before the various sections of the College are subsequently published in full in medical periodicals.

The papers listed in the following pages may be found by consulting the *Index Medicus*, provided a sufficient length of time has elapsed from the date of publication to the next succeeding quarterly number of the *Index Medicus*.—[Editor.]

PROCEEDINGS

OF THE

SECTION ON OPHTHALMOLOGY

JANUARY, 1931 TO DECEMBER, 1931

JANUARY 15

Dr. George H. Cross reported the case of a man, aged fifty-four years, who had been referred for the purpose of reconstruction of his left socket, which was so shrunken and cicatrized that it was no longer possible to wear an artificial eye.

At examination there was found a solid object, deep in the orbit. Upon questioning, the patient stated that thirty-four years before, following the loss of his eye, a Mules' operation had been performed. In order to enlarge the socket at this time an incision was made through the tissues of the orbit and a hollow glass sphere about 13 mm. in diameter was removed. Strangely, this sphere was filled with a slightly turbid, bloody fluid which had evidently seeped through a pinhole opening. Where the fluid came from was an unsolved problem.

During the thirty-four years this glass ball was *in situ* there had been gradual absorption of the glass, leaving a very thin spot which had evidently broken through. It was thought, on account of the length of time, this would be of interest to the Section, especially so on account of the newer operations used in enucleation.

Large Penetrating Wound of Lower Lid and Eyeball

Dr. Frederick Krauss presented a case of a large, penetrating wound of the lower lid and the eyeball, with recovery of vision.

The patient was struck with a large piece of glass from an exploding rootbeer bottle which he had placed near a stove. The cut extended nearly the whole length of the lower lid. The cut in the eyeball began about 5 mm. below the ciliary body, at about

six o'clock, and extended about 12 mm. down and back. The eyeball was very soft. The vision was *nil*. The vitreous was filled with blood; therefore, no view of the eyeground was obtainable though the anterior media remained clear. After suturing the wounds the patient was put to rest with atropin and a solution of metaphen for antisepsis. After ten days the vitreous began to clear. A Vossius ring was present. Through the retina a broad white tear could be seen in the periphery extending halfway to the macula, but lateral to the same. The retinal edges of the cut were bloodstained throughout. The vitreous has cleared except for a dense, organized mass below and very far anterior. The cut in the retina has apparently healed and is seen with difficulty. The field of vision is but slightly cut. The central vision with correcting lens is 20/30. Externally there is a slightly depressed, pigmented scar in the sclera at the site of the injury. The Vossius ring, though less marked, has persisted.

Discussion. Dr. Zentmayer asked whether the Vossius ring had been examined with the biomicroscope for the purpose of determining whether the lesion was pigment or blood, a question which is still in dispute.

He said he should like to cite an experience that he had some years before while doing an advancement operation. As he placed one of the scleral sutures he was conscious of the sudden change in resistance and thought the needle had probably gone through the sclera. Shortly afterward a line of blood appeared on the anterior surface of the iris, just sufficient to obscure vision. Two days after the operation he examined the eye with the ophthalmoscope and found that the patient had a typical Vossius-ring cataract. Dr. Holloway confirmed the diagnosis.

This patient had been under observation for some time in the clinic and had been refracted several times, and the media had always been noted as clear.

Dr. Zentmayer further stated that Hesse endeavored to produce a Vossius-ring cataract by injecting a small amount of blood into the anterior chamber, but because of the rapid coagulation of the blood he was successful in only one instance. After learning of Dr. Zentmayer's experience, Finnoff undertook to cause experimentally this lesion by producing an intraocular hemorrhage. He produced in one case a partial ring. He, however, expressed the belief that rabbits were not altogether suitable for the experiment.

In reply to Dr. Zentmayer, Dr. Krauss said that the anterior chamber had always been free of blood. He had examined it under the slitlamp and there were very fine punctate opacities which he had interpreted as being pigment rather than blood. However, he said he was not the expert that Dr. Zentmayer was with the slitlamp, and that he would be very glad to have him study the case further if he cared to do so, but his impression was that it was pigment.

Homonymous Hemianopsia in Diabetes

Dr. deSchweinitz reported briefly the case histories of 5 patients with diabetes, 2 men and 3 women, and homonymous lateral hemianopsia. He referred to 18 other cases in the literature, and especially to those collected by Wilbrand and Saenger.

He pointed out that this field defect was not due directly to the diabetes, but was caused by vascular lesions in the occipital lobes.

The hemianopsia, right or left lateral, may be at once complete; may begin as a quadrant defect, followed later by loss of the homonymous lateral fields or with scotomas which subsequently expand into complete hemianopsia or lateral homonymous scotomas remain unchanged.

In 1 case, reported by Wilbrand, the field defect presented itself in the form of the so-called macular hemianopsia. Occasionally the hemianopic fault entirely disappears; usually it is permanent; it may be detectable only if the field is tested under reduced illumination, especially if partial cataract is present. The hemianopsia may be the only symptom of the brain lesion, or may be followed by others, for instance, hemiplegia. In all cases that had come to autopsy, as reported in the literature, the lesions were vascular (hemorrhagic) in character.

Discussion. Dr. Holloway stated that he was delighted that Dr. deSchweinitz had brought this subject before the Section. It was quite true that hemianopsia associated with diabetes was not mentioned in connection with this affection in many of the treatises concerning this subject. As far as his own experience was concerned, he had seen but two instances, one in a patient who was seen at the University Hospital many years ago, and the second in a private patient. He felt, as he knew was the case with Dr. deSchweinitz, that the condition was doubtless the result of vascular changes so frequently seen in connection with this disease.

Cysts of the Orbit

Dr. Frederick Krauss presented the history of 2 cases of cyst of the orbit. The first case was in a woman, aged seventy-six years, who presented a dense tumor in the upper inner angle of the orbit, slow in growth and associated with no pain. Careful palpation indicated a soft center in the tumor. The *x*-rays were negative. Nasal symptoms were absent. Upon dissection, the tumor was found to be filled with very thick mucoid substance, the culture of which showed staphylococci only. A second collection

of the same material was found in the deeper ethmoidal cells when the curette was carried through a small carious opening in the os planum. The periosteum had been elevated by the secretion to make a cavity extending to near the optic nerve entrance. Rapid healing occurred after a large opening had been made into the nose, with primary closure of the wound.

In the second case the tumor was associated with edema of the lids and considerable proptosis of the left eyeball. The *x*-ray disdisclosed disease of the frontal ethmoidal and antral sinuses of that side. The middle turbinate did not appear diseased, but the outer nasal wall was slightly edematous and pressing the turbinate. In this case the intranasal operation was performed, with the evacuation of large quantities of an offensive pus from the ethmofrontal region. A complete exenteration of the ethmoids was performed, with washing of the antrum, which also contained offensive mucopus. The external operation was not performed as the condition improved with complete disappearance of the exophthalmos.

The Treatment of Interstitial Keratitis

Dr. Joseph V. Klauder and (by invitation) Dr. Harold F. Robertson, presented an analysis of 105 case records of interstitial keratitis, with reference to the following data: Age of patient: Up to five years, 6 patients; six to ten years, 24; eleven to fifteen years, 29; sixteen to twenty years, 21; twenty-one to twenty-five years, 15; twenty-six to thirty years, 8; thirty-one to thirty-five years, 1; thirty-five to forty years, 1. Total: females, 63; males, 42.

The absence or presence of the major stigmata of congenital syphilis concern: Facies: Present in 39, absent in 37, suggestive in 6, no mention in 23. Teeth: Hutchinsonian in 16, abnormal in 35, negative in 54. Deafness: Present in 4, absent in 101. Sabre tibia: Present in 20, suspicious in 6, absent in 62, no mention in 17. Clutton's joints: Present in 18, suspicious in 9, absent in 78. Wassermann and Meinieke reaction: Positive in all but 4.

From the foregoing it will be seen that about one-third and less of the entire series presented objective evidence of congenital syphilis, whereas all but 4 patients presented positive Wassermann reactions. The Meinieke was positive in 2 of these 4.

Involvement of eyes: Both eyes showed active involvement in 35, unilateral involvement in 37, inactive in 33.

Period of observation: About one-third of the patients attended the clinic for about six months, one-third for about one year and one-third from one year to thirty-four months.

Results of treatment: Of 37 patients with an initial attack of interstitial keratitis in one eye there was an involvement of the second eye in 10. In 3 of these, however, this involvement occurred after very little treatment, then a lapse of all treatment owing to lack of attendance; in 5, after moderate treatment, then a lapse of all treatment; in 2, involvement of the second eye occurred after thorough treatment. In the entire series recurrence occurred in one or both eyes in 3 patients after moderate amount of treatment.

Treatment employed was made of the following drugs: neoarsphenamine, sulpharsphenamine (intramuscularly and intravenously), tryparsamide, unctions of mercury, iodides (orally and intravenously), bismuth (intravenously and intramuscularly), bismarsen (arsenical bismuth preparation), means to increase the general state of health of the patient. One or more of these drugs were employed in different patients in order to determine the most effective means in treatment of interstitial keratitis. Of the foregoing drugs, bismuth therapy appeared to yield more favorable results than any of the other drugs. Moreover, bismuth is much less toxic than mercury or arsenic. It is well tolerated by children and its administration is much more practical than that of the arsenicals.

The importance of early diagnosis and early treatment were emphasized. It was advocated that an injection of bismuth be given the first day the patient is seen, even though the diagnosis is not definitely established, since such injection is harmless.

The more favorable formula of treatment appeared to be a weekly injection of bismuth intravenously (10 mg. of colloidal bismuth), a weekly injection of bismuth intramuscularly, and from 15 to 25 grains (depending on the age) of sodium iodide. The dosage of bismuth for intramuscular injections advised at different ages is as follows: 25 mg. of the metal bismuth, up to the age of five years; 50 mg., from five to six years; 75 mg., from six to seven and a half years; 100 mg., from seven and a half to ten years; 150 mg., from ten to fourteen years; 200 mg., over fourteen years.

Lantern slides were used, showing the evolution of interstitial keratitis with and without treatment, and unusual features of the disease.

The phenomenon of the involvement of the other eye was discussed. It was pointed out that such involvement while the patient is receiving treatment is unique in the entire field of syphilology. There is only one other clinical circumstance, and that is rare, in which a comparable process occurs. That is the occurrence of cutaneous syphilides while antisyphilitic therapy is being administered. In these circumstances the administration of another antisyphilitic drug (changing from an arsenical to mercury or bismuth) invariably causes the involution of the cutaneous syphilide.

The rôle of anaphylaxis as a cause of sensitization of the uninvolved eye was discussed.

The possibility of antisyphilitic drugs not reaching the cornea was also discussed. Recent studies by others, however, show that some drugs do reach the cornea. Experimental studies are being conducted to determine if antisyphilitic drugs, injected into rabbits, reach the cornea.

Syphilis was visualized as an infection from a clinical and laboratory viewpoint. It was pointed out that the mechanism of immunity in syphilis is largely a tissue reaction rather than humoral. In experimental rabbit syphilis the incidence of eye lesions can be varied by using experimental means to vary general tissue reactions. It appears from the foregoing visualization that the cornea responds feebly to a syphilitic process.

Unsuspected Brawny Scleritis in a Case of Retinal Detachment With Secondary Glaucoma

Dr. T. B. Holloway and (by invitation) Dr. W. E. Fry reported the history of a man, aged forty-nine years, who had been having exacerbations of pain and redness of the left eye, extending over a period of six months. There was no history of injuries or previous inflammatory attacks. The blood Wassermann was positive. The vision of the right eye was 5/5 and the left, light perception.

Right eye, negative. Left eye: the pupil was dilated and fixed. There was a diffuse bulbar injection with marked overfilling of the episcleral vessels. The cornea was clear; anterior chamber shallow. By oblique illumination one could readily discern an enormous detachment of the retina. By transillumination a questionable shadow was obtained centrally in the pupillary area, but it was not regarded as dependent upon a growth. With Schiotz tonometer the tension of the right eye was 16, of the left 33.

Microscopic examination showed the characteristic findings of the brawny scleritis. Dr. Verhoeff was good enough to examine the sections and reported that he believed the condition to be as above described.

In closing, Dr. Holloway stated that this patient had been sent to his office for consultation, and that with the scant light perception, the detachment of the retina and secondary glaucoma he felt there was but one decision to make, and that, despite the fact that no definite shadow, dependent upon a probable growth, was present. He stated that a brawny scleritis was not even thought of at the time the patient was seen, and that the eye did not manifest any evidences of the clinical findings usually associated with this affection. He stated that the literature showed several instances where the condition was unsuspected and the diagnosis had been made after enucleation.

FEBRUARY 19

Congenital Cataract With Unusual Visual Acuity

Dr. Zentmayer presented a case of congenital cataract with unusual visual acuity and pseudo-accommodation, thirteen years after operation. This patient when a girl, aged nine years, was referred to him by Dr. R. T. M. Donnelly, in May, 1917. The history of the case leaves it uncertain as to the type of cataract originally present. After an illness of several weeks the patient accidentally discovered that she had very poor sight in the left eye. Previous to the illness she attended school and had no trouble with her eyes.

When first examined there were diffuse opacities throughout both lenses. By November, 1917, the left lens was opaque throughout. Two needlings and a linear extraction were made. In January, 1918, under atropine, vision with a $+12 = +125°$ c ax. 165, was 5/7.5. In March, 1918, without atropine, with a +12, it was 6/6 pt. She read type 0.50 at 10 cm. with distance glasses.

The right lens was needled but once, in January, 1919. In April, with a +13, vision was 6/6 and she read type 0.75 with difficulty with this glass.

In December, 1920, vision in the right eye having fallen to 1/120, the Ziegler V-shaped capsulotomy was done. Following this, vision with $+10 = +2.50$ c ax. 105, was 6/5.

The conditions which pertain today are:

O.D. +10 = +1.75 *c* ax. 100° 6/4 5′ card.
Dr. Cowan's card 6/5. Part.
O.S. +10 = +1.50 *c* ax. 70° 6/3 5′ card.
Dr. Cowan's card 6.5. One mistake.

There is orthophoria and esophoria for distance, and 7° of exophoria at 33 cm. There is bar reading, and full binocular vision, including Verhoeff's cerebral binocular test.

With distant correction and the eye wide open:

O.D. Type 1 pp. 17 cm.
O.S. Type 0.50 pp. 13 cm.

With distant correction and nipped lids:

O.D. Type 0.50 pp. 13 cm.
O.S. Type 0.37 pp. 11 cm.

Without glasses:

O.S. Type 1 pp. 14 cm.

The addition of a −0.25 sph. to her distance glass reduces her vision almost a line.

Ophthalmoscopic Examination. O.D. (with a +16): Pupil almost round. A fine synechia at 12 o'clock. A hairline of capsule which shows along the nasal border of the iris; the pupil seems otherwise clear. Disk vertically oval, with a clear-cut edge. A cilioretinal vessel to the outside. O.S.: Pupil round. A crescent of dense white capsule, beginning at 1 o'clock and extending around the nasal and lower border of the pupil to 4 o'clock, with a vacuolated rim of tissue between this and the pupillary center. Beyond on the temporal side the pupil is perfectly clear. The disk is rather dimly, but the rest of the fundus plainly, seen.

Biomicroscopic Examination. (Made by Dr. Alfred Cowan.) O.D.: Vitreous bulging forward through the pupillary space. This is covered by an extremely thin, transparent membrane. During contraction of the pupil the curvature of this vitreous front increases, but it is unlikely that this would aid materially in her ability to see near objects. The accommodative effect seems to be due almost entirely to partial closure of the lids. O.S.: The pupillary space contains a dense white sheet of remaining capsule in which there is a tiny opening. This most probably acts as a stenopeic aperture. The evidence of this is borne out by her ability to see clearly at certain times without her glasses.

Case of Spontaneous Cyst of the Iris

Dr. Edward A. Shumway reported a woman, aged fifty-six years, who had been operated upon for carcinoma of the rectum. The cyst had appeared within two years and produced no discomfort or disturbance of vision. There was no history of traumatism or operation. The cyst was transparent, the anterior wall being thin and crossed by fibers representing the iris stroma; the posterior wall was in contact with the pigment layer of the iris. The cyst had forced the lower part of the iris from its ciliary border, and was in contact with the cornea below, without producing any cloudiness of that structure.

Dr. Shumway discussed the theories as to the origin of spontaneous iris cysts in the anterior part of the iris in distinction from those developing from the epithelium of the posterior layer of the iris and those arising from an attachment of the ciliary processes to the posterior iris surface (Tertsch's classification), all of which are to be distinguished from the more common cysts which arise from ingrowth of epithelium into the anterior chamber after traumatism or operation.

In view of the complication of a tumor of the rectum the eye had been searched carefully for metastasis, but none could be found. Dr. Shumway had decided not to remove the cyst by operation, because of the patient's age, the complicating inoperable carcinoma and the absence of ocular discomfort.

Discussion. Dr. Reese said that some time ago a case of this type appeared at the Wills Hospital and that Dr. Cowan made a diagnosis with the slitlamp of cyst of the iris. Dr. Reese had felt that it was a tumor. There was no history of injury, as he recalled it, and the cyst differed from Dr. Shumway's in that the color was consistent with that of the iris throughout. There was very definite elevation of the cornea at the site of the cyst, which was practically in the same situation as in this case. He made an attempt to remove it and got into difficulty, discovering the cornea to be abnormally thin, so much so that he desisted and placed a conjunctival flap over the whole mass and the eye recovered uneventfully.

He asked Dr. Shumway whether there was bulging and thinning of the cornea at the site of this cyst.

In reply to Dr. Reese, Dr. Shumway said that, so far as he knew, thinning of the cornea had not been reported in the cases examined anatomically, but opacification of the cornea was common. In operating it was necessary to remove the entire cyst with the part of the iris involved, to prevent recurrence. With simple puncture the cyst would promptly fill up.

Melanosis of Anterior Segment of Globe With Elevations on Iris

Dr. H. Maxwell Langdon presented a paper upon melanosis of the anterior segment of the globe with irregular elevations on the iris. He said that melanosis of various parts of the body is of fairly frequent occurrence in the form of moles, nevi or plaques. It occurs mostly in brunettes and possibly has some relation to the formation of malignant tumors, as Coats found 7 instances of melanotic sarcoma in 26 cases of diffuse melanosis of the uvea. Heine reported nevus and sarcoma in juxtaposition in the iris, with evidence of transition from the benign to the malignant form, though Fuchs thought the change from nevus to sarcoma in the iris had not been shown with any certainty, and felt that melanosis is merely an incident of sarcomatous growths.

In the eye melanosis is usually unilateral and varies a great deal in its extent from a few pigmented spots in the iris to a general pigmentation of lids, sclera and uveal tract.

The condition was very thoroughly described by Bourquin, and in March, 1930, Goldstein and Wexler made a most exhaustive study of a case in Mt. Sinai Hospital, in New York, with the microscopic report of one of the eyes. This case was associated with neurofibromatosis, many pigmented skin moles and acromegaly.

The eyes showed hyperpigmentation of the uveal tract, the posterior pigment layer being almost double the normal thickness, with protrusion at the pupillary margin. Irregular masses on the anterior surface of the iris, with hyperplasia of the anterior iris endothelium and irregular pigment masses through the stroma. The pigment excess was also in the ciliary body, including its process, and in the pectinate ligament. There was no evidence of round-cell infiltration, new-vessel formation or karyokinesis.

The source of this pigmentation in the iris is difficult to explain. Fuchs objected to its being called pigmented nevus owing to the absence of nevus cells; this places the source among the pigmented fixed tissue cells. These may be the endothelial ectodermal cells, or the chromatophores of the stroma, which are believed by most authorities to be mesodermal in origin, and this seems to be the most likely source.

Reese believed this to be the source of the pigment in his case of melanosis, and Treacher Collins believes these mesodermal cells

have a phagocytic property, taking up melanin from degenerating epithelial cells.

This condition is, of course, one entirely different from that of blue sclerotic, where the coloration is due to the normal choroidal pigment showing through the abnormally thin sclera.

The case which is here reported is that of J. DeL., a boy, aged three and a half years, the third child in a family of four children, the oldest thirteen years and the youngest one month, none of the others being similarly affected, nor are the parents. The condition was noticed first about two months ago, but has probably been there for a much longer time.

The pigment involves a ring of sclera about from 4 to 6 mm. out from the limbus and the iris, the lids and the rest of the uveal tract being quite normal. The right eye has a brownish-gray iris and seems normal in all respects. As near as can be told in a child of this age, the function of each eye is normal. The affected sclera looks as though it has been peppered with pigment granules and this description also fairly well applies to the iris. This structure in daylight is a dark sooty-brown with granules thickly peppered over its whole surface, the crypts entirely filled in and the overlying pigment so thick that one has a feeling of not looking at the iris itself, but at the pigment on the iris.

The iris in this case, as in Friedenwald's and in several other cases, showed irregular protuberances or wartlike growths over its surface. George Coats used the term "golf-ball" iris in describing a case of his. In Goldstein's case the iris was the site of multiple melanotic nodules. In the case here reported the melanin does not seem to be in the nodules, but on them, except for their topmost parts, which under the corneal microscope seem to be free of pigmentation and to resemble normal iris tissue.

This is well shown in a sketch which Dr. Cowan was good enough to make for me under the corneal microscope, the illumination of which naturally imparts a brighter tone to the pigment than does daylight. The uveal tract seems to be quite free from excess pigmentation in all other parts.

Discussion. Dr. deSchweinitz, who had had the opportunity of examining Dr. Langdon's and Dr. Friedenwald's patient (a girl, aged about sixteen years), expressed the opinion that the lesions were probably identical in nature, although not exactly similar in manifestation.

The refractive error of Dr. Friedenwald's patient was a mixed astigma-

tism; the visual acuity was markedly improved when the patient tilted her head toward either shoulder and gazed obliquely. In these circumstances a constant lateral nystagmus, ordinarily present, practically ceased.

Dr. Cowan said the thing that struck him most in this case was the gradually diminishing size of the elevations from the pupil toward the periphery of the iris. Those nearest the pupillary border were best developed. They appeared as spherical elevations with tops of fuzzy material that did not look like iris tissue and certainly was not pigment. It was nearly colorless. These elevations decreased in size until at the periphery they were like tiny tips. The regular distribution over the surface of the iris and the decrease in size and maturity from the pupil outward were remarkable.

Dr. Langdon (in closing) said that in reference to the subnormal visual acuity in Dr. Friedenwald's case, that in most of those cases that have been reported there was no change in the visual function at all. In Goldstein's case there was decided loss of vision and difficulty in studying the condition during life. This patient died in the hospital and the eye was secured for section and a very complete study was made.

The case shown tonight has apparently normal visual acuity, but this boy is so young that it is hard to tell accurately.

A Note on Radiational Cataract

Drs. deSchweinitz and Baer, describing radiational cataract as one which is caused by any form of radiation which is "absorbed in quantity by the lens," made reference to the three chief varieties of this lesion, namely, those due to exposure to infrared rays (glassblower's, etc., cataract), to ultraviolet rays (rather uncommon) and to radium and *x*-ray emanations (comparatively fairly frequent). But they confined their remarks chiefly to that form which may be caused by radium when used as a therapeutic agent for the destruction of an epibulbar sarcoma, illustrated by the record of a case history.

The cataract may be regarded as a special type and in most instances begins at the posterior cortex in an oval or disklike form, rather granular in appearance and containing fragments of crystallized lens protein. Beneath the anterior capsule a delicate layer of opacity may form; also vacuoles. The lens structure between the anterior and the posterior cortex is at first unaffected.

They referred to the observations of a number of authors (for instance, Rohrschneider, Schnyder, Meesmann and Zentmayer), and their examinations confirmed those made by these authors, not only when the agent was radium, but also *x*-rays. The char-

acter of the cataract is similar, no matter which form of radiation causes it.

Their patient's eye exhibited another complication, namely, paresis of the outer half of the iris adjacent to the area originally occupied by the sarcoma.

They also referred to the invariable development of epibulbar conjunctival telangiectasis as the ultimate result of the radiation—a quite characteristic phenomenon, as originally pointed out by Birch-Hirschfeld.

They also described the development of quiet iritis in one patient successfully treated for epibulbar sarcoma by radium. In this connection they made reference to Flaschentrager's observation, quoted by Koby, of vascular lesions (dilatation, thickening, cleavage of walls) in the ciliary body of the eye of a patient who had been irradiated for cancer of the breast.

Such being the case, similar vascular lesions in their patient might account for the iritis, which, however, rather rapidly subsided under the influence of the usual treatment.

Their patients had been successfully treated by means of radium emanations by Drs. Pancoast and Pendergrass, of the X-ray Department of the University Hospital. The patients are always warned that a sharp reaction may follow the radiation and that some permanent disturbance of vision may result.

Discussion. Dr. Pendergrass said that the occurrence of telangiectasis in the conjunctiva following irradiation is usually an evidence of over-radiation. Irradiation usually causes an endarteritis obliterans of the bloodvessels supplying the part, and thereby destroys the normal nutrition of the treated area, and in an effort to overcome this Nature forms new bloodvessels. In the treatment of melanotic lesions of the cornea the tendency is to overirradiate rather than underirradiate, because of the seriousness of the prognosis.

In the cases reported by Dr. deSchweinitz and Dr. Baer this type of irradiation was used. Some of the rays were penetrating and some were superficial. Recently we have been employing superficial irradiation so as to prevent a possible deep effect.

It is a little difficult to explain the formation of a cataract such as that described by Dr. deSchweinitz. If the cataract was formed following a disturbance in nutrition of the bloodvessels of the ciliary body, one could easily understand how irradiation might produce it. I have been told that the disturbance of the blood supply of the ciliary body would not cause changes in the posterior cortex of the lens. If this is true, then one

would have to explain the effect in some other way, and a possible explanation is the following:

Absorption of the superficial rays would cause changes that would subsequently lead to telangiectasis. The more penetrating, or gamma rays, might be absorbed in the region of the posterior cortex of the lens and account for the changes observed by Dr. deSchweinitz and Dr. Baer. This explanation is theoretically possible, but is probably less tenable than the changes produced in the posterior cortex secondary to the disturbance in the blood supply of the ciliary body.

Dr. Shumway said that he thought the changes in the lens might be due to the disturbances in the vessels of the ciliary body and consequent interference with the nutrition of the posterior part of the lens, just as occurs in disease of the uveal tract in the posterior part of the eye. There are no cells lining the posterior capsule of the lens such as are present on the anterior capsule, and proliferative cell changes, therefore, could not be produced by radium in this position.

Localizing Tears in Retinal Detachment

Dr. Alfred Cowan and (by invitation) Dr. L. F. McAndrews described a method of accurately localizing tears in retinal detachment. The patient is seated at ½ meter distance from a tangent screen and is told to rotate his eye laterally, fixing a point about 50 degrees from the center of the screen. The examiner then locates the lesion with an ophthalmoscope. An assistant, with a meter stick or a piece of string, projects the line of direction, found by the examiner, back to the tangent screen. The angle found by the examiner must be corrected to allow for the bending of the rays of light. This is done by deducting one-fourth from the angle, both in the vertical and the horizontal meridians. This corrected point is now placed on the tangent screen.

To find the corresponding point on the sclera, one can use a scaled perimetric chart. The chart represents the entire retina projected on to a flat surface, its center corresponding to the fovea, and the 90-degree circle to the ora serrata. On this chart are vertical and horizontal lines, drawn 4 degrees apart. Each of these 4 degrees square is equivalent to 1 mm. on the retina. The point found on the tangent screen is transferred to this perimetric chart. Considering that the 90-degree circle corresponds to the ora serrata, and that the latter is 8 mm. behind the limbus, the correct distance of the tear from the limbus can be quickly and accurately estimated.

Discussion. Dr. Peter said that the plan submitted by Dr. Cowan and Dr. McAndrews for localizing tears in detachment of the retina is

perhaps less complicated than the majority of the other methods which have been offered to us. There is no difficulty whatever in locating the tear with reference to the corneal meridian. The difficulty in all cases rests in being able to properly determine the depth of the tear.

A fact which all of these methods cannot definitely determine is the location of the tear when the fluid is withdrawn and the retina is again in contact with the sclera. It matters little how accurate a method can be in locating the tear while the fluid is in place; none of the methods can tell one definitely where the tear will be when the cautery is applied.

I have performed a good many Gonin operations, and have been fortunate enough to strike the tear in a great majority of cases; but it is only by careful observation with the ophthalmoscope and by shrewd guessing that one can even approximate the location of the tear. I find, therefore, that good judgment is important.

If one will remember that the average disk is about 1½ mm. in diameter, the size of the tear can be approximated with reference to the size of the disk, and by careful measuring in disk diameters from the disk forward to the tear, and remembering the size of the cornea and the distance of the ora serrata from the limbus, the location can be approximately determined without the use of any one of the many complicated methods which have been suggested.

It should also be remembered that the position of the tear in the retina when the patient is in an upright position is not the same as when the patient is in the recumbent position, as we find him on the operating table. Any method which is employed should be practised with the patient in the recumbent position.

MARCH 19

A Case of Bitemporal Paracentral Scotoma

Dr. Shannon reported a case of bitemporal paracentral scotoma in a man aged thirty-six years. Following serum injections of typhoid, dysentery and cholera, all in one day, in September, 1929, he had suffered from blurred vision and severe pain in his eyes. A diagnosis of glaucoma had been made and he had been given a weak solution of eserin for constant use.

Examination of his eyes revealed no abnormalities of the anterior segment. Tension by tenometer, 20 mm. Hg in the right eye and 19 mm. Hg in the left eye. The ophthalmoscopic examination was negative.

With correcting glasses a vision of 6/6+ was obtained in each eye.

Form and color fields were within normal limits.

A small bitemporal paracentral scotoma was mapped out just temporal to the point of fixation in each eye. Fields taken at varying intervals for four months showed no change in the findings.

Special Examinations. Urinalysis negative; blood Wassermann and spinal fluid negative; examination of blood showed a white count of 8800, otherwise negative; *x*-ray of teeth revealed several bad teeth and these were removed; *x*-ray of skull by Dr. Manges was negative.

The diagnosis of small vascular lesions of the chiasm of toxic origin seemed justifiable. Luetic diseases of the chiasm, which is frequently found in these bitemporal lesions, was discarded by reason of the negative blood Wassermann and spinal fluid. Pituitary disease was eliminated, as the *x*-ray of the sella turcica failed to note any abnormalities, and, further, there was no evidence of increase in size of the scotomata.

By elimination, it was concluded that the small vascular lesions were due to focal infection, abscessed teeth or, more probably, the result of the serum injections.

Discussion. Dr. deSchweinitz expressed the opinion that the bitemporal hemianopic scotomas depicted in the visual field charts of Dr. Shannon's patient were best explained by the presence of a small lesion in the chiasm at the position which the essayist had described, due to vascular obstruction, exactly as had been pointed out by Wilbrand and Saenger and by Traquair.

The former authors believe that such a condition is due to syphilis; but Traquair, in his reference to this matter, states that the vascular obstruction is "usually of syphilitic origin," and therefore he does not exclude other etiologic factors.

In the present instance syphilis as a causative agent has been eliminated by the history and by all the usual tests. Hence it seems likely that this isolated chiasmal lesion may be ascribed to a localized vascular disturbance caused by the violent reaction which followed the various inoculations to which this patient submitted.

The visual field defects exactly correspond to those which Wilbrand and Saenger have described, and are likely to be permanent. The macula has been "spared" and the visual acuity is not interfered with.

Dr. Zentmayer said he would like to report a case in which there was binasal paracentral scotomata in association with hemichromatopsia. The patient, a woman, was seen at the Wills Hospital, in 1889 or 1890, and observed over a period of nineteen years. She was aged twenty-seven years, quite stout, married and had two children. She had had some symptoms that suggested pituitary trouble with disturbance of central vision such as Dr. deSchweinitz has described in these cases.

At first the field was full for form, but there was a bitemporal hemichromatopsia. The field and vision remained unchanged for a long while, but later a bitemporal hemianopsia for form as well as color was present. She was examined a great number of times, and ten years later developed binasal paracentral scotomata, larger than the ones described by Dr. Shannon. The case was extremely interesting, and in view of the pituitary symptoms, this being before the day of the *x*-ray, Dr. Spiller thought that she probably had a growth.

At the end of fifteen years she regained her temporal field in part and central vision was improved from 6/15 to 6/9. The paracentral scotomata remained. She died of pneumonia and no postmortem was held.

The only explanation seems to be that it was a lesion of the chiasm and the growth had broken through the sella turcica, relieving pressure, and in that way the physiologic block was lifted.

Nonglaucomatous Halos

Drs. John T. Carpenter and Chapin Carpenter presented a paper upon this subject in which they said that halo vision is a prominent symptom of glaucoma. Its occurrence when glaucoma is absent is, therefore, of interest and there are not many references in ophthalmic literature to the nonglaucomatous halos.

Their differentiation is important, even though halos are infrequent as sole indication of glaucoma. Col. Elliot and Treacher Collins have laid emphasis on this point. Nonglaucomatous halos may be seen around lights by certain individuals, consisting of diffraction rings. These are also present when mydriatic has been instilled, and are due to the endothelial layers of the cornea. Brundenill Carter reported similar phenomena in his own eyes, due to changes in the lens. Other halos are seen normally after caustic applications to the lids or when mucus is present in the conjunctival sac. Fogged spectacles also produce them and they have been present in iritis. Incipient changes in the lens cause halo vision, particularly nuclear cataract due to the sclerosis. These halos were persistent in the cases reported by us.

Nonglaucomatous halos are smaller and less vivid than glaucomatous ones. Their size varies from 4 to 8 degrees.

Elliot suggests a test, originated by Ensley, in which, if a halo is seen around a candle placed 10 feet away, a card passed vertically in front of the pupil, if glaucoma is present, will, as it crosses the pupil, only reduce the intensity of the halo. In halo due to cataract the opposite portions of the halo disappear entirely. The explanation of this is that the corneal edema in glaucoma is more

or less uniform and through any part of the cornea the halo would be seen, while in lens changes such is not the case.

Glaucomatous halos vary in occurrence, depending on the presence or absence of corneal change, while those due to incipient changes in lens are constant.

Three cases were observed where incipient cataracts of the nuclear type were present, and where there was halo vision persistently noted by the patients. This did not vary, and in all the cases the tension was found normal with tonometer readings, fields normal and the test suggested by Elliot proved the halos to be nonglaucomatous. Two patients were observed over a year without any alteration in the condition except advancing lens changes, but without any glaucomatous changes in the nerves or visual fields.

The Hazards of Antifreeze Methanol

Max Trumper, Ph.D. (by invitation), presented a paper upon this subject. He said that automobilists, and especially those working in service stations and in garages, should know of a danger to which they may be exposed from the presence of wood alcohol used as an antifreeze mixture in radiators of cars and trucks. Especially at this time of the year when, owing to the warmer weather, more of this wood alcohol will evaporate from the radiators, the danger of being poisoned by its vapors is greater than usual. Many do not realize that antifreeze methanol, or methyl alcohol, is the chemical name for wood alcohol. The containers of methanol, sold to the public under many trade names, are not always labeled as poisonous. Much is sold from barrels bearing no conspicuous poison label to protect the public. Automobilists have been further misled by labeling the product as a "completely denaturized alcohol" or as a "188-proof alcohol," omitting the word "wood." The coining of a variety of trade names is an old method of obscuring the presence of concentrated wood alcohol, the knowledge of which would impair its sale.

It is important to remember that both the new synthetic methanol and the old crude methanol are poisonous. (The difference is that the latter when evaporated from radiators contained obnoxious warning odors.) The new synthetic product has no odor and, therefore, the automobilist receives no warning when it is evaporating and he is breathing it. This danger is also a serious one for those individuals who use methanol as fuel for their alcohol lamps

or chafing dishes (a use which some dealers have recommended in order to increase sales). Methanol is quite as poisonous when inhaled into the lungs as when taken into the stomach.

Thus far we have considered the dangers inherent in wood alcohol or methanol by reason of its new uses in which it may be breathed into the lungs or gain entrance by way of the skin. We must not overlook the dangers involved from its misuse, that is, its being used as a drink. The U. S. Bureau of Mines recommends that "all antifreeze methanol be brightly colored" to avoid such misuse. This recommendation, in my opinion, will hardly be followed by all producers until legislation requires it of them. Were methanol obtainable only in drug stores, it would be promptly included under the laws governing the sale of all poisons, especially because it has a taste and odor somewhat similar to grain alcohol. In this connection it is encouraging to note an opinion rendered by the Attorney-General of the state of Ohio, which reads: "Synthetic methyl alcohol may be sold in Ohio only by registered pharmacists." Even should we not go this far; I do believe it important to warn the general automobile-riding public to be on guard against the vapors of wood alcohol.

Discussion. Dr. E. A. Shumway said that Dr. Trumper's paper was timely, as it called attention to the fact that methanol was a methyl alcohol preparation; but he doubted if its use in automobile radiators would be dangerous, as the fumes would not get inside the cars to such an extent as to make much contamination of the air. Drs. deSchweinitz, Tyson and others have reported serious disturbance of sight in workmen shellacking the interior of large vats, and Dr. Tyson has reported cases in which women had been affected while varnishing lead pencils. In all instances methyl alcohol, or so-called Columbian spirits, was present in the varnish, but lack of ventilation in the vats or rooms was an important factor. A number of cases were on record in which carbon monoxide poisoning occurred in closed cars in the winter season, from the escape of the gas into the closed sedans, from leaky exhausts or defective heaters, but he could not see that this could occur from a solution in the radiator.

Dr. deSchweinitz, after briefly referring to his own observations and others reported in the literature on methyl alcohol blindness due to inhalation aided by absorption through the cutaneous surfaces, pointed out that, so far as he was aware, in all these instances the subjects of this toxemia had worked where ventilation was entirely lacking, and he agreed with Dr. Shumway that in the presence of proper ventilation it was most unlikely that visual disturbances due to inhalation would occur.

He referred to the researches of Ward Holden and Birch-Hirschfeld, who had attributed methyl alcohol blindness to nutritive changes in the ganglion

cells of the retina. On the other hand, the retinal ganglion cells of dogs acutely and chronically poisoned by the administration of commercial methyl alcohol, during a rather elaborate research carried on at the University of Pennsylvania by Fewell, De Long and himself, failed to show even the slightest departure from normal. Similar negative results were obtained by Jonas Friedenwald and Felty, in Baltimore. Also, in all these experiments there had been at no time any evidence of blindness or ophthalmoscopic change. In Schwarzkopf's experiments the animals exhibited no ophthalmoscopic lesions, but microscopically degenerated ganglion cells were found.

An effort to explain these contradictory results has been made by assuming that they depended on the kind of methyl alcohol employed—chemically pure methyl alcohol, commercial alcohol, etc., or the deleterious influence has been attributed to an admixture of fusel oil.

Henry Smyth, at the Laboratory of Hygiene of the University of Pennsylvania, however, failed to find any fusel oil in any of the methanols he used in his experiments, which were performed on guinea-pigs whose retinas exhibited definite ganglion-cell changes.

The late Dr. Harold Gifford attributed the blindness to a simultaneous action of wood alcohol on the ganglion cells and the optic nerve, and Jonas Friedenwald has recently stated that this agent acts on the whole optic nerve.

Casey Wood and the late Dr. Frank Buller regarded methyl alcohol intoxication in human beings as a typical example of idiosyncrasy. Whether a similar idiosyncrasy exists among animals he was quite unprepared to discuss or maintain. Evidently the whole subject remains as an interesting field for further research.

Dr. Shumway added that the effect of methyl alcohol on the system was produced by the intermediate products of its decomposition before it was finally oxidized. Ethyl alcohol was immediately reduced to CO_2 and water, in the body, but methyl alcohol was first converted into formaldehyde and formic acid, which poisoned the organism, the one by interfering with the oxidizing efforts of the cells, and the other by withdrawing oxygen from them. It had been shown, moreover, that the previous ingestion of ethyl alcohol still further prolonged the removal of the formic acid from the body, delaying at times the appearance of the visual disturbance and the fatal issue.

APRIL 16

Spontaneous Complete Absorption of a Cataractous Lens

T. B. Holloway, M.D., and Alfred Cowan, M.D. report: J. P., aged forty-six years, a mechanic, married, came to the Dispensary of the University Hospital on October 24, 1928, complaining of dimness of vision in the left eye for the past two weeks. He had

been unaware of poor vision previous to this time, although he admitted that he had had no eye examination. There was no knowledge of traumatism. Outside of a gastric ulcer, for which he was operated upon, in 1922, he had always been in good health. He had been married twenty years and had no children. He was the oldest of 18 children, of whom 13 died in infancy or early childhood; otherwise his family history was of no importance.

The pupils were equal and reacted promptly to the various reflexes. In the right eye the vision was 6/6. The media was clear. The disk was healthy, oval and with the upper border slightly blurred. The vessels were somewhat tortuous and the veins showed some "kinking" where the arteries crossed, indicating early sclerosis. There were no fundus lesions.

In the left eye vision was reduced to light perception. The cornea and aqueous were clear. The undilated pupil was blocked by a cataractous lens. With dilated pupil the whole lens appeared shrunken, especially in the anteroposterior diameter, making it waferlike. When the patient fixed straight ahead the lens was slightly luxated down and out, leaving a very small, crescentic, dark area in the upper nasal portion of the pupillary space. Physical examination and all laboratory tests revealed nothing abnormal. The patient again denied any knowledge of injury to the eye.

He was seen at intervals of about two weeks. The lens slowly but steadily decreased in bulk until, in April, 1929, it was reduced to a thin wafer, which sank almost to the lower pupillary border. A good view of the fundus could be obtained. Absorption was now progressing more rapidly, and in May, 1929, the small, white disk that represented the remains of the lens was visible only when it bobbed up with marked rotation of the eye. A flat, perfectly transparent, thin membrane across the whole pupillary space, just behind and free from the iris, could be seen with the slitlamp and corneal microscope. This membrane was not lens capsule, although the surface had a very fine, shagreen appearance and gave a bright iridescence in specular reflection. Numerous tiny white spots were scattered over the surface of this membrane, and behind it, in the vitreous, were two narrow, transparent, scroll-like structures. One of these extended from 8 to 10 o'clock and the other extended from 1 to 4 o'clock in the pupil, 8 mm. in diameter. In December, 1929, no trace whatever of any part of the crystalline lens could be seen.

On April 7, 1931, the delicate scrolls in the vitreous could still be seen. The thin, transparent membrane, stretching across the pupillary space, lay just behind the plane of the iris. Behind the membrane there was an optically clear space, and behind this space, in the vitreous, were the tubular structures. With the ophthalmoscope these structures appeared transparent. A few fine opacities could be seen in the vitreous. The disk was slightly oval and healthy. There was some pigment roughing around the periphery of the fundus, and in the extreme temporal periphery a circumscribed retinochoroidal lesion, measuring about 4 mm. in diameter, with pigment processes, one of which was longer than the others and extended toward the disk along a retinal vessel. The form field showed some nasal contraction. The vision was 6/6 with +14, +0.25 ax. 45.

The intraocular tension had always been normal, but on April 9, 1931, it was found to be 36 in the right eye and 33 in the left eye (Schiotz). The patient was given a 0.5 per cent solution of pilocarpin, and on April 13, 1931, the tension was 33 in the right eye and 30 in the left eye. At no time were there any other symptoms of glaucoma.

Discussion. Dr. Holloway said that they had watched this case with a great deal of interest at the University Hospital, for the past few years, and that all of the details were as Dr. Cowan had described. The interesting thing, of course, about this case was the spontaneous absorption. He said that they all knew that in many of these cases of spontaneous absorption one of the interesting phases was the frequency with which it was associated with glaucomatous symptoms. While a certain number may have these symptoms, no active signs were present in this patient.

There was no history of injury to the eye. The patient has been seen at regular intervals and there has been a slow absorption until at present there is no trace remaining. There is a delicate, practically structureless membrane across the pupillary space, but this membrane is definitely not lens capsule. There are a few fine opacities in the vitreous.

This week the tension, which has been normal, showed evidence of a slight rise, but without any pain or congestive symptoms whatever. At one place in the periphery a delicate pigmentation can be noted overlying a retinal vessel for a short distance.

Dr. Posey said that, in 1918, he was consulted by a boy, at that time fourteen years of age, for glasses, and at that time nothing abnormal was detected in the right eye other than the fact that vision could not be brought up to more than 5/12 with correcting lens.

There was no history of traumatism, and when seen the eye was quiet, the lens clear and the pupil the same size as its fellow and the pupillary

border normal; the iris reacted well to light. He was seen again in 1921 and no change in the eye was noted. The boy was seen again in 1923, when there were some opacities noted in the lens, and at that time the boy was turned over to Dr. Posey's assistant, Dr. Swindells.

In 1929 Dr. Posey was again asked by the father of the patient to see the boy, as he felt that something should be done, when it was found that a cataract had developed on the right eye. There had been no inflammatory symptoms and no rise of tension. In February, 1930, the eye showed a fully developed cataract, the anterior chamber was normal in size and the iris reacted to light.

The question naturally was as to the cause of the cataract. It was thought possible that there might have been a foreign body going through the lens, but *x*-ray study showed no foreign body. It was then thought possible that there might be a growth of the ciliary body which pressed upon the lens and interfered with its nutrition.

Dr. Posey counseled against operation in view of the uncertainty of the origin of the cataract and as what might be back of it. Shortly after this the boy began to notice that he could see out of that eye and he again consulted Dr. Swindells, who called Dr. John T. Carpenter in consultation, when it was found that the lens was partly subluxated and that there was some vision eccentrically, *i. e.*, counting of fingers when held down and out. The eye was quiet. Dr. Posey saw the boy again about a month ago when the vision of the eye had become disturbing, as there was diplopia, the visual axes not being parallel. The anterior chamber was now found to be deep, the lens shrunken and the pupil practically clear. With a +9 D. the vision came up to 5/15. There has been practically total absorption of the cataract, probably explained by a previous rupture of the posterior capsule of the lens by some blunt force in early life which had left no discernible injury, no evidence of damage to the anterior capsule having been visible at any time.

Dr. Zentmayer said that in November, 1890, he had seen in his office a man, aged sixty-two years, a typesetter, who came on account of an inflammation in the right eye, and gave a history that the sight of his left eye began to fail in 1876. This progressed until at the end of three years he was unable to see more than light. In 1887 he had been told at the Pennsylvania Hospital that he had a cataract and should have it removed.

When first seen by Dr. Zentmayer the uncorrected vision was 3/150, the sight having gradually improved, and with a +11 it was 5/5, with a 4′ card. In the right eye there was an active iritis and the lens was cataractous. In the left eye the media were clear, the disk appeared small and there was a broad, atrophic conus to the temporal side. The fundus was plainly seen with +11 D. The eye was aphakic. When last seen, five years later, the vision in this eye was 5/6.

Dr. Zentmayer said that, of course, his case was not as authentic as that of Dr. Holloway and Dr. Cowan, as he did not observe the absorption of the lens, but only saw the end-result. He felt quite certain, however, that traumatism was not a factor.

On another occasion he had observed the absorption of a cataractous lens. He had seen the child in infancy with the persistence of a hyaloid vessel. The lens at that time was clear. When the child was five years of age the lens became cataractous. When seen after the lapse of several years the lens matter had become entirely absorbed and the hyaloid artery could again be seen extending from the posterior capsule to the disk.

Regarding the development of increased tension in the eye, in his experience this occurs so frequently in aphakic eyes as a late sequel to cataract extraction, that he had felt the need of some means of preventing it. This, he thought, might be accomplished by doing a preliminary iridectomy, making it basal, as you would for glaucoma, hoping that this might prove a prophylactic measure. He had done this for only about ten years, of course too brief a time to judge whether it had been of value.

Senile Macular Changes of the Retina Associated With Focal Infection

Dr. Hunter Scarlett said that a review of the literature indicates that macular changes may be associated with focal infections variously distributed throughout the body. Four case histories were given, each with a diagnosis of senile macular degeneration.

The associated focal infection in the first case was an apical abscess. The second and third had diseased tonsils, while the fourth presented a prostatic involvement with a possible dental infection. All these cases had marked retinal arteriosclerosis, also vitreous opacities.

The dental *x*-ray examination of the first case was negative, but since the patient had a devitalized tooth on the side where the macular lesion was present, it was extracted, with a resulting discovery of hemolytic and nonhemolytic streptococci in culture. Vaccines were given and vision in the right eye, which had dropped from normal to 6/20, returned to 6/10. One of the tonsil cases, after having been treated with the electric cautery, showed a diminution in vision. In conclusion Dr. Scarlett said:

First, given an eye with markedly diseased retinal vessels, plus a focal infection liberating toxins or bacteria into the bloodstream, that it was reasonable to expect changes similar to those seen in senile macular degeneration.

Second, that vaccines are of aid where focal infections are present.

The Syphilitic Optic Atrophies

Dr. Joseph Earle Moore, of Baltimore, Md. (by invitation), discussed the rôle of syphilis in the etiology of the optic neuropathies and emphasized the necessity for careful and complete study of

each patient as a diagnostic problem. The most important of the syphilitic optic atrophies is the so-called primary atrophy. Its relationship to tabes and to other forms of neurosyphilis was mentioned, and it was estimated that at any one time there are about 50,000 patients in the United States with this condition. Great emphasis was laid on the necessity for early diagnosis by means of coöperation between ophthalmologists and syphilologist, the routine ophthalmologic examination of all neurosyphilitic patients and use of such diagnostic aids as carefully taken visual fields and tests of dark adaptation.

The pathology of the lesion was discussed and it was pointed out that the process begins in the marginal fibers of the intracranial portion of the optic nerves, anterior to the chiasm; but that there is as yet no agreement as to the relationship of degenerative to inflammatory processes or as to the presence or absence of treponemas in the optic nerves.

The treatment of syphilitic primary optic atrophy, to the introduction of the arsphenamines, was hopeless, and even these drugs, routinely employed, did not materially improve the prognosis. Two modern treatment methods, namely, subdural therapy and fever therapy, have been found in various parts of the world to be of some value in arresting the progress of the disease and in preserving useful vision. (Lantern slides were shown illustrating the application of these methods of treatment in individual patients, followed over periods of three to fourteen years.) It was concluded that in a patient with early primary optic atrophy (vision in the better eye 20/40 or more), subdural treatment by various routes (intraspinal or intracisternal) and with various substances (arsphenaminized serum, neoarsphenamine, mercury or air), combined with appropriate general antisyphilitic treatment, offered at least a 50 per cent chance of arrest or even slight improvement, and that by this means useful vision might be preserved for periods of time long exceeding the average expectancy of two years from the onset of symptoms Fever therapy (malaria), while also successful in a somewhat smaller proportion of cases, is more likely suddenly to extinguish the patient's remaining vision. It is more dangerous to life, and patients treated with it have not yet been followed for a long enough period of time to insure permanency of results. The choice of a treatment method depends on the general physical and neurologic status of the patient, as well as on the condition of his eyes.

Discussion. Dr. Stokes, speaking as a syphilologist, said that he felt that Dr. Moore had made, on the basis of the evidence submitted, a genuine and hopeful contribution to the still very limited control of primary optic atrophy in neurosyphilis. The situation is of interest particularly because throughout the larger part of the field of neurosyphilology intraspinal therapy is steadily losing ground and is being replaced by tryparsamide and the various devices for inducing high body temperatures. In fact, though when he first came to Philadelphia he equipped his department at the University Hospital for the administration of intraspinal therapy, he doubted if it had been necessary to treat a dozen patients by this method in the past six years. This, of course, was largely because this form of treatment was not applied to primary optic atrophy. Dr. Stokes said they would await with keen interest Dr. Moore's further and confirmatory report on the very much larger material of this type which he is handling at the present time. He said that in the meanwhile he very much feared that the individual isolated observer will continue to have the same disconcerting experiences with individual cases that have discouraged them all in the past. Dr. Stokes said that only in larger aggregates, and by persistent patient determination, can one bring forward such impressive evidence for a relative degree of hopefulness in prognosis in a condition both logically and practically unamenable to treatment. It would seem that services such as that of the Wills Eye Hospital, in which accomplished syphilologists, like Dr. Klauder, have at their command what must be a very large material, are the proper places, in Philadelphia particularly, for putting this new hope to the test of experience.

OCTOBER 15

Blepharoplastic and Ptosis Operative Procedure

[Dr. Vilray P. Blair, St. Louis, Mo. (by invitation), gave an illustrated lecture on blepharoplastic and ptosis operative procedures. Numerous slides and moving pictures of the various operative procedures were shown.—Ed.]

NOVEMBER 19

Left Plastic Iritis Which Does Not Respond to Mydriatic and Parenteral Protein Injections

Dr. William F. Bonner exhibited a case of iritis in an infant, aged four months, with a history of failure of left pupil to react to light; the baby was always otherwise healthy. The mother gave a history of septicemia following childbirth. The blood count and blood Wassermann was negative.

A Case of Bilateral Optic Atrophy Due to Methyl Alcohol Poisoning

Dr. Shannon presented a case of bilateral optic atrophy due to methyl alcohol poisoning. The history briefly is as follows:

Mr. H., aged twenty-one years, while visiting a garage, drank one-half glass of what was supposed to be gin, on Thursday afternoon, May 1, 1931. Two hours later he suffered from acute gastritis, with vomiting and severe pains in the abdomen. On the following morning he again vomited on four separate occasions, and on the same evening and through the night he vomited at frequent intervals.

On Saturday morning, thirty-six hours after the drinking of the gin, he noticed for the first time that his vision was affected. He was then rushed out to the hospital and within a short time following his arrival he noted a marked depreciation of vision. Shortly afterward he went completely blind. There has been no return of vision since. He visited my office on September 21, 1931, about four months later, and my examination revealed the following:

Vision: No light perception in either eye. Pupils: Widely dilated, unresponsive to light. Rotation of eyeballs normal.

Ophthalmoscope: O.D. Media clear; disk white, with deep glaucomatous-like excavation. The arteries were narrowed. The macula and periphery of the fundus exhibit no abnormalities of note. O.S. Same.

Tension (as measured by Schiotz tenometer) was 15 mm. Hg in the right eye and 18 mm. Hg in the left.

It was found impossible to secure any of the gin at the garage for an analysis, but by reason of the severe symptoms shortly following the ingestion of the gin by the patient it seems reasonably safe to conclude that it must have contained methyl alcohol. Curiously three of his companions imbibed as freely as he did: one suffered abdominal distress, but in none of them was the vision affected. The interesting feature of the fundus picture is the extraordinary excavation of the disk.

Dr. deSchweinitz quotes Schnabel (in the discussion of glaucomatous excavation) as stating that the excavation is due rather to a degenerative process than from pressure. While it is the consensus of opinion that pressure is the fundamental cause of excavation in glaucoma, may we not perhaps look upon the excavation in

this case as of a cavernous type due to degenerative changes in the papilla?

This young man had always been in excellent health. His present appearance, his age and tension (as recorded by the tenometer) would exclude glaucoma as an etiologic factor.

The symptoms of methyl alcohol poisoning (gastrointestinal disturbance accompanied by abdominal pain, general weakness, nausea, vertigo, headache, with dilated pupils and blindness) were definite in this case and therefore confirm our diagnosis.

As we know, improvement in the vision is noted in some cases, with subsequent relapse into permanent blindness. This did not occur in this patient.

We must remember, in methyl alcohol poisoning some persons are largely immune as far as permanent damage to the organisms is concerned. Only one of the four who drank the gin was seriously affected. Wood has stated that "If ten persons drink, say, 4 ounces of wood alcohol within three hours, all will have marked abdominal distress and probably four will die, two of them becoming blind before death.

The exact manner in which methyl alcohol acts upon the internal organs is unknown, although once absorbed from the stomach or lungs into the blood, it is not so easily eliminated and burned up as ethyl alcohol. Apparently small and frequent doses tend to a cumulative effect in the system, resulting in serious damage to digestive, sight and nervous systems. According to Wood, there is a destructive inflammation of the optic nerve fibers or retinal elements (or both), followed by atrophy; however, in this patient the atrophy would suggest a primary form of a distinctly degenerative character.

Discussion. Dr. Zentmayer said that Dr. Percy Friedenberg, many years ago, published his observations on the optic nerve head in methyl alcohol amaurosis, in which he described the condition present in the case shown by Dr. Shannon. In addition to the excavation, he found in one case an increase of glial tissue in the bottom of the cup, but in all the other instances there was an extrapapillary overgrowth of connective tissue.

There is a difference of opinion as to the primary effect of the alcohol, whether it be on the ganglion cells of the retina or whether it be an effect on the nerve fibers or the interstitial substance. Jonas Friedenwald's experimental work indicates that the whole nerve is involved. A research fellow of the Rockefeller Foundation has stated that if animals poisoned with methyl alcohol are kept in the dark blindness does not supervene.

Dr. deSchweinitz stated that he considered this a very highly interesting paper, and that he had also noted in the subjects of methyl alcohol blindness excavation of the nerve head simulating a glaucoma-cup, originally described by Percy Friedenberg, and had reported one such case, but had never observed this condition so perfectly developed as is evident in Dr. Shannon's patient.

He referred to the fact that the amount of wood alcohol capable of producing blindness varied considerably; 2 to 5 drams have caused blindness, as occurred in one of his own patients, while recovery after drinking ½ pint, or even larger quantities, has been observed. In short, methyl alcohol intoxication is a typical example of idiosyncrasy.

The deadly effects of this poisoning may be due not only to drinking it, but also to inhalation and cutaneous absorption, as has been experimentally demonstrated. The blindness has been attributed to nutritional changes in the retinal ganglion cells (Ward Holden, Birch-Hirschfeld, Henry Smythe and others here and abroad), but their experimental results were not confirmed by other investigators (Jonas Friedenwald and A. Felty, in Baltimore; Fewell and Perce De Long, at the University of Pennsylvania). It is possible that the poison attacks the whole of the nerve.

The toxemia has also been attributed, he said, not to the methanol itself, but to adulterants (for instance, fusel oil, etc.), and to imperfect combustion of the alcohol in the system and the production of formaldehyde. But Reid Hunt's analyses have demonstrated that the chief toxicity belongs to the methyl alcohol. The whole subject from the experimental standpoint should be reviewed.

Dr. Holloway said that in his opinion, if these cases be taken as a group, idiosyncrasy was not the chief factor, but more important was the question of how much had been ingested and absorbed. Generally you will find that those who have vomited freely, or when the stomach has been relieved of its contents soon after ingestion, do not suffer to the same extent as do those who retain the poison.

While idiosyncrasy is doubtless a factor, the same is true in tobacco and ethyl alcohol poisoning, but doubtless not to the same extent. Dr. deSchweinitz has referred to the breaking-down of the alcohol into other toxic substances, and it is my belief, when we definitely know the terminal products responsible for these symptoms, we will be able to show whether the process begins in the ganglion cells, the nerve fibers or the interstitial tissue. I referred to the treatment of these cases in 1923, before the Section on Ophthalmology of the American Medical Association.

DECEMBER 17

Removal of Oxidized Tissue Remaining After Foreign Body of Cornea (A Handle for Corneal Burrs)

Dr. George H. Cross read a paper on this subject in which he said that corneal burrs are used to remove the brownish-stained, burned tissue which remains in the cornea following the removal

of hot metallic foreign bodies. They also are used to remove the undercut edge of a foreign body crater so annoying to the patient, often causing him to return in a short time complaining that all the foreign body was not removed. This edge provides an excellent harbor for bacteria that may produce ulcers. The burrs are useful to smooth out the roughened surface area from which a foreign body was removed. Originally burrs were soldered in a metal handle. This was too expensive to throw away.

This handle for corneal burrs (manufactured by George P. Pilling & Sons, in Philadelphia) provides a satisfactory way to hold the burrs and permits quick changing from one size to another as the case may require. The burrs are made in different sizes, are inexpensive and may be purchased from instrument or dental supply firms and will fit this handle.

These corneal burrs are very easy to operate. Place the burr on the area to be removed and twirl the handle a few times between the thumb and fingers; only light pressure is required.

Lupus Erythematosus of the Conjunctiva and Eyelids

Dr. Joseph V. Klauder and Dr. Perce De Long presented a paper on this subject in which they said that lupus erythematosus of the conjunctiva is an extremely rare condition. English and American authors who make mention of it do not describe the disease. The few cases that have been reported have been by foreign writers. Description of the disease as reported by others was discussed. The appearance of the disease in the patients shown at the meeting was essentially the same as that described by others.

Three patients, two women and one man, with lupus erythematosus of the conjunctiva were shown. In two the conjunctival lesion was previously diagnosed trachoma. The clinical picture, however, did not present the characteristics of trachoma, in that neither showed any granulations or follicles. In one patient there was an extreme redness of the conjunctiva, with a velvet-like edema involving the exposed surface of the conjunctiva of the upper lids. The involvement was more pronounced on one eye. The patient complained of photophobia and a mucoid discharge. In the second patient the appearance of both lower lids was similar to the foregoing; in addition there was an involvement of lupus erythematosus of the lids and lid margins. The third patient showed edema of the

conjunctiva of one eye, with a sharply defined violaceous patch and an additional well-defined depressed area which was dry, bluish-white and atrophic in appearance.

The diagnosis of lupus erythematous of the conjunctiva was suggested by the finding of typical lesions of the disease elsewhere on all of the patients. The clinical features of the disease as it is seen on the skin were briefly discussed. The characteristic features of lupus erythematosus on the skin can be correlated with conjunctival involvement with reference to the violaceous color, edema, sharp margination of the lesion and, later, the appearance of atrophy.

The appearance of lupus erythematosus on the eyelids is essentially the same as the disease on the skin. On the lid margins it superficially simulates blepharitis. The following features are characteristic: The lid margins are dry, not moist, and covered with finely adherent scales; the color is somewhat violaceous, not red and inflammatory; there is no matting of the eyelids. Later, the margins are atrophic, their surfaces irregular, the violaceous color disappears, and the cilia are permanently destroyed. Since atrophy caused by lupus erythematosus is not of a contractile nature, ectropion and eversion of the lid margins are not likely to occur.

The patients were treated with gold and sodium thiosulphate, which is effectively employed in the treatment of lupus erythematosus. Such treatment caused either complete or almost complete disappearance of the various lesions of the disease at the time of presentation. Treatment was being continued.

Lymphomas of the Conjunctiva

Dr. C. E. G. Shannon and (by invitation) Dr. L. F. McAndrews presented a report of a case of bilateral lymphomas of the conjunctiva. The patient, a woman, aged fifty-six years, noticed small masses appearing beneath the bulbar conjunctiva, near the superior rectus muscle insertion of each eye. These masses had been present for about one year. The mass in the left eye gradually increased in size until it had involved the upper half of the globe. On the right eye they remained stationary. The general physical and blood examinations were normal. The diagnosis was made by microscopic examination. Radium was used to remove the growths. Four applications were given with excellent results. This is the first case reported in which radium was used to remove conjunctival

lymphomas. A complete review of this subject was also presented. At the present time there is no satisfactory explanation as to the etiology of lymphomas of the orbit.

Discussion: Dr. B. L. Crawford said that lymphomas of the conjunctiva have a very characteristic histologic picture. The lymphomas in this location seem to be more definitely neoplastic than in other locations of the body. The diagnosis of lymphoma is frequently made in lesions of other parts of the body where there is a more or less indefinite collection of lymphoid type of cells without true tumor formation. This neoplasm, as illustrated by the lantern slide, is composed of a fairly uniform type of cell which closely resembles the lymphoid cell, rather loosely arranged, with little stroma and numerous small bloodvessels.

In differentiating the lymphomas from round-cell sarcomas there are several points to be considered, as follows: Malignant tumors are rarely multiple, as in this instance; the uniformity of the type of cell and the lack of irregularity of the cells and the absence of mitosis of the nuclei usually seen in malignant tumor; the type of bloodvessels in the stroma, and the presence of a fairly definite capsule. Leukemic infiltration is excluded by a careful examination of the blood.

Dr. Zentmayer asked whether the bloodvessels in lymphomas were not different from those in a malignant tumor? To which Dr. Crawford replied that the small bloodvessels in the stroma of the lymphoma had well-defined walls and were not in such an intimate relation with the tumor cells as in a sarcoma.

Dr. Zentmayer said that through the courtesy of Dr. Shannon he had seen this patient before treatment was started. With the history and a negative physical and blood examination, he had suggested the possibility of its being a case of amyloid degeneration of the conjunctiva. He said he had no apology to offer, as in the pathological report in one of the cases hyalin degeneration was noted as a feature.

Dr. Crawford had pointed out the difficulty in the histological differential diagnosis between such growths and small-cell sarcoma.

Dr. Shannon (in closing) said that he wished to thank Dr. Crawford for his interesting discussion.

The three points of interest, in his opinion, were: (1) Its rarity—about thirty cases have been reported in which blood and histologic studies had been made; (2) the rapid disappearance of the growth following the application of radium; (3) the fact that while radium was applied to the left side only, in the early treatment of the case both eyes responded equally well. He had no explanation to offer for this phenomenon.

The Recession Operation in Squint

Dr. Luther C. Peter presented a paper upon this subject. He said that uncontrolled tenotomy has given way to the more rational recession suture, which is a safeguard against subsequent convergence insufficiency and the tendency to paralytic squint in the

opposite direction. The operation of Jameson dates from 1922. Rae reported a recession suture at the International Congress of Ophthalmology in 1929. The new operation which I am about to report differs in many respects from the Jameson and the Rae operations. The several steps in the operation were devised without any knowledge of the Jameson method. The important steps consist of the following:

1. A vertical incision of the conjunctiva 1 mm. back of the point of insertion.

2. Subconjunctival dissection toward the canthus.

3. Introduction of a tendon hook, with slight freeing of the muscle insertion from the capsule.

4. A double-armed whip-stitch introduced near the upper and lower borders close to the muscle hook, from without in, so as to grasp the muscle properly and spread it upon the sclera.

5. Cutting of the tendon, leaving 1 mm. of stump.

6. 2-mm. bites in the superficial sclera, widely separated, so as to spread the tendon on the sclera, and precisely placed so as to avoid torsion.

7. Passing of the suture through the center of the tendon stump, 3 mm. apart, and through the conjunctiva anterior to the stump.

8. The suture is tied with a loop, after drawing up the tendon to its proper place.

9. Closing of the conjunctiva and sclera by a running untied suture. The conjunctival suture is removed on the fifth day, and the scleral suture on the seventh.

The essential difference between this and the Jameson suture lies in the use of No. 1 silk instead of catgut, which admits a very fine needle and consequently offers less trauma to the sclera. All the strain of the suture is placed upon the tendonous stump. Closure of the suture by means of a loop admits of modification on the second or third day if necessary.

The indications for the operation, as compared with the shortening operations on the weak muscle, are briefly stated as follows:

In the correction of moderate degrees of squint advancement of the long muscle should be given the preference on the basis of sound underlying surgical principles. In squint of high degrees, or in such instances in which more than one surgical procedure becomes necessary, the recession suture logically becomes a supplementary operation of much value.

In esophoria and exophoria the choice of operations is largely

determined by the ratio of abduction to adduction. When abduction is excessively low it is wise to increase the power of the external rectus. Conversely, if adduction is excessive and abduction is normal a recession of the internal rectus is indicated.

Discussion. Dr. Reese said that he thought this paper very timely and that he would like to induce some of the members of the Section to do this operation. He had been doing it for the past ten years and it had worked out very well. He felt it an advantage to select a technique which required only one scleral stitch, as there have been several unfortunate cases reported of perforation of the globe. There is slight danger of this if one is careful to place the stitch so superficially that the needle can be seen passing through the sclera. This is facilitated if moderate pressure is made on the globe.

Dr. Peter (in closing) thanked Dr. Reese for his courtesy and added that he was perfectly right that one need have no fear of perforation of the sclera by the needle. The needle should be a fully curved, very small needle, and the point should be visible at all times, and in this way there is no danger of perforation of the sclera.

The Ophthalmological Importance of Focal Infective Prostatitis.

Dr. P. S. Pelouze (by invitation) presented a paper upon this subject. He said the extent to which the various focal infective eye conditions may be influenced by infections of the prostate gland is not generally recognized. While many of these eye conditions are due originally to dental, tonsillar or sinus infections, they rather commonly fail to be cured after attention to these foci because there is an overlooked infection in the prostate.

Comparatively few cases of focal infective prostatitis have any direct or remote relation to gonorrhea. They are predominantly secondary to tonsillar or dental infections and cannot be cured permanently so long as these causal foci persist. At least 35 per cent of all males beyond thirty-five years have an infected prostate gland. At least 72 per cent of all males exhibiting focal infective symptoms have a prostatic infection; 69 per cent of these have infected tonsils and 61 per cent have infected tooth roots. Further, 64 per cent of those who have teeth extracted for infection have other infected teeth within two years. It, therefore, is obvious that assurance that there are no dental foci can only be temporary. Consequently, every patient who has had such an infection should have repeated roentgenograms of the teeth during the succeeding two years, at least.

Even gentle massage of such a prostate gland usually will cause an increase in the ocular symptoms. If such a reaction does not follow one of the first few massages of the gland in all probability there is no relation between the eye lesion and the prostatic infection. These increases in ocular symptoms following prostatic massage are analogous to a vaccine reaction and are due to the forcing into the system of quantities of toxin in excess of the ocular toxin tolerance.

Under such circumstances it is obvious that prostatic massage must be thought of in terms of toxin dosage and the pressure varied according to the severity of the reaction produced by the previous treatment. Great damage can be done to the eye by rough prostatic manipulations, for which reason few treatment procedures require more gentleness and judgment. These prostatic massages should not be repeated more often than twice a week and, if the eye condition is growing worse because of them, they should be discontinued until it improves or reaches a stationary stage.

Recurrences of the ocular lesion are more likely to be due to other focal infections, but they make wise another prostatic study. It is common for prostatic reinfection to recur as the result of later dental infection and this may prevent cure of the eye lesion despite attention to the teeth.

The diagnosis of prostatic infection is made by the finding of pus in the prostatic secretion. It cannot be made by rectal palpation alone.

Discussion: Dr. deSchwenitiz referred to some of his experiences with nonspecific prostatitis as an etiologic factor in ocular disorders, iritis and certain forms of relapsing keratitis, published some years ago, and to Dr. Swinburne's contention that these forms of prostatitis should be regarded equally with chronic infection of the tonsil, and to Dr. Zentmayer's excellent paper on this subject.

He expressed his satisfaction that Dr. Pelouze had brought the results of his large experience to the attention of the Section. He differed somewhat with the essayist's opinion as to gonococcal iritis, which is a perfectly well-established form of iridocyclitis, chiefly plastic in type, which may be associated with or follow an arthritis, due to the influence of gonococci (or perhaps their toxins) on the anterior uveal tract; indeed, in these circumstances the gonococci have been recovered from the anterior chamber by Sidley-Huguenin. This form of iridocyclitis is favorably influenced by the administration of large doses of gonococcic vaccine (Neisser bacteria), as has been demonstrated in clinical reports, published here and abroad.

Dr. Zentmayer stated that, in 1926, he presented a paper on "The Prostate as a Remote Focus of Infection in Ocular Inflammations," before

the Section on Ophthalmology of the American Medical Association. His object in so doing "was to direct attention anew to the importance of searching further than the mouth, nose and throat for the portal of infection in cases of metastatic infection, and to emphasize that this is not infrequently to be found in the prostate gland.

At the time he presented this paper the only other two papers on this subject in ophthalmic literature were one by Thomasson and that of Dernehl. In the paper the points brought out by Dr. Pelouze, of the danger of manipulation of the prostate during the time when metastatic inflammation is active, and that the prostatic infection is more often non-gonococcal than gonococcal, were emphasized.

Sarcoma of the Choroid

Dr. T. B. Holloway brought before the Section a patient with sarcoma of the choroid. A male, aged twenty-three years, had just come under observation. He gave a history of having his right eye struck by a hammer three years ago. This was not followed by any inflammatory reaction and the eye remained quiet for a year. Then the vision of this eye became impaired and has grown steadily worse. Upon seeking medical advice he was advised that he had a detachment of the retina.

Extraocularly the eye was negative except that the direct light reflex of the right eye was slightly less than the left, and the indirect light reflex of the latter possibly slightly less than that of the right. There were a few vitreous opacities. Beginning about half disc diameter from the temporal side of the disc, there was a large round and well-circumscribed solid detachment of the retina that extended over the macular region and far toward the periphery of the eye ground. At the extreme limit outward one could note a small disc-sized choroidal erosion just below the horizontal meridian. Deep vessels over the summit of the growth were rather conspicuous and in several places small, vague areas of pigmentation were seen. The apex of the detachment could be clearly seen with a +20 D lens.

Dr. Holloway stated that he thought the conditions present showed a typical picture of a sarcoma. The field showed a slight contraction with practically a complete loss of the whole inferior nasal quadrant and extending to the blind spot. Careful tests were made to see whether any difference could be shown in the field corresponding to the detachment over the growth and where it sloped off its margin. A definite change could not be interpreted. Needless to say, enucleation had been advised. The left eye was negative.

PROCEEDINGS

OF THE

SECTION ON OTOLOGY AND LARYNGOLOGY

JANUARY 21

Roentgenographic Anatomy and Physiology of the Larynx and Adjacent Structures

Dr. Henry K. Pancoast: The upper respiratory tract is becoming an important field for roentgenological diagnosis. The essential factor is a careful study of the soft structures of the neck. Fluoroscopy and roentgenography are equally important in the examination. The former shows functional movements of the larynx and all adjacent structures, and derangements of function, as well as defects in outline, encroachments on lumen and displacements due to growths, paralyses and inflammatory conditions, while the latter records these findings and shows finer details.

For fluoroscopy the eyes must be so well accommodated as to see the ventricle of the larynx and movements of the arytenoids. The normal ventricular air space is "zeppelin-shaped." If seen intact and clearly, one can exclude a growth or inflammatory condition of the larynx and recurrent laryngeal paralysis. If not seen distinctly, one of these three groups of conditions must be present. The arytenoid movement must be studied, with phonation of vowels and with swallowing. Having lateral and sagittal movements, they must be observed in various directions. The freedom of movement of the epiglottis and larynx as a whole must next be watched. Freedom of movement of the soft palate is best determinded by having the patient whistle. The swallowing act should be studied next, with a thin bismuth suspension. This shows the motor mechanism of the pharynx, larynx and upper esophagus, the movability of the epiglottis, arytenoids and larynx; it outlines growths on the tongue, epiglottis and in the pyriform sinuses, and determines the patulousness of the food passages.

We have been able to observe and record the following sequence of events in the swallowing act: As the mouth fills and is filled, the tongue moves backward to close off the oro- and nasopharynx. At the next step several things happen simultaneously and quickly. The larynx rises so that the arytenoids come in contact with the under surface of epiglottis, which has been tilted slightly back by the tongue and held there. This closes off the larynx. The tongue pushes the bolus into the pharynx or allows it to drop into the latter. The tongue then tightly closes off the oro- and nasopharynx and the pharynx contracts and forces the bolus into the esophagus. When all food has entered the latter, and the pharynx and pyriform sinuses are empty, the larynx descends, the tongue goes forward, the epiglottis springs back into its vertical position and all structures have returned to their normal positions.

Interference with the normal swallowing act in any way, and with the movements of the arytenoids, and the absence of the ventricular air space are observations which indicate a careful endoscopic examination, especially when the roentgenograms do not show the exact lesion present. In vocal cord paralysis there may be complete absence of the space; it may be a narrow half-zeppelin shape or the lower posterior portion may be obliterated. The mechanism of acquired speech following laryngectomy has been studied and recorded by fluoroscopic and roentgenographic study.

The normal soft-tissue space between the pharynx, larynx and trachea and the cervical vertebral bodies has been carefully measured for different ages in each sex, and increased thickness is known to indicate growths, abscess and foreign bodies in the upper esophagus, as the case may be.

The Value of Roentgen Diagnosis in Diseases of the Neck and Upper Respiratory Tract. (Lantern Slide Demonstration)

Dr. Eugene P. Pendergrass: The fluoroscopic and roentgen study of the neck and upper respiratory tract is one of the most valuable supplementary examinations the laryngologists and bronchoscopists have at their disposal. The roentgen examination offers a permanent record of the location and extent of the disease or growth, just as the microscopic slide becomes a permanent record of the pathological process. An investigation of the upper respiratory tract should include a collective study of the pharynx, neck

and chest. In infants and children this wide inclusion should be adopted as a routine procedure in every chest examination. Many suspected intrathoracic conditions may be explained in part, or entirely, by obstructive lesions high up.

Our interests in, and realization of, the importance of the aid of the roentgen diagnosis in this particular region began with the study of a few cases of retrolaryngeal abscess. Appreciation of the diagnostic possibilities became a reality after studying the observations of Hay.

Foreign bodies in the upper respiratory tract are clearly visible when lodged in any of the air spaces, because, no matter what the nature or atomic weight, the intruder is surrounded by air and is easily visible. It may be necessary to use opaque solutions or substances to demonstrate nonopaque foreign bodies in the upper end of the esophagus. Nasopharyngeal growths and growths in the posterior nares are easily detected because of their encroachment on the relatively small space in the nasopharynx.

Lesions involving the soft palate either cause a swelling of this organ or a disturbance in innervation or both. Carcinoma of the tongue, lingual thyroid, the vallecula, either with or without involvement of the epiglottis, cause a disturbance in the swallowing act and a deformation of these structures which can be very easily detected.

In infections of the larynx the roentgenologic evidence is absence of the ventricles. Fluoroscopically one may note a disturbance in innervation and motility of the arytenoids. Acute diphtheria may cause a distinct encroachment on the lumen of the larynx and upper end of the trachea, due to the membrane. This particular disease usually involves the conus elasticus. Tuberculosis may cause an obliteration of the ventricles and disturbance in the motility of the arytenoids. The motility of the arytenoids is particularly observed during the swallowing act.

Stenosis of the trachea may be due to infections or trauma or may be of congenital origin. The most frequent type of stenosis of the trachea is that found following diphtheria. The ventricle of the larynx is clearly visible in all normal individuals during phonation and is "zeppelin-shaped." It occupies the anterior three-fifths of the larynx. Its presence is of distinct value in the diagnosis of infections, growths and recurrent paralysis. All of these conditions may cause an obliteration of the ventricle.

In growths the ventricle may be encroached upon or it may be entirely obliterated. Extensive growths may cause a disturbance of the upper end of the air spaces in the trachea. This disturbance in outline has various formations, depending upon the type of growth.

Retropharyngeal abscess causes a distinct decrease in the retropharyngeal space and is easily demonstrated at the fluoroscopic and roentgenological examination. It is important to remember that in children two phases of respiration are necessary in arriving at a definite diagnosis.

Discussion. Dr. Willis F. Manges: This work represents the type of roentgenology that requires a roentgenologist. Some physicians do not care for a roentgenological report, but merely request the films. In work of this kind so much depends upon technique that even roentgenologists who have not studied such work as that done by Drs. Pancoast and Pendergrass will find themselves unable to make diagnoses. There is nothing to criticize. Commendation is all that is appropriate. In studying the larynx and adjacent structures I have found it difficult to decide whether shadow variations represent normal variations or pathological variations. As Drs. Pancoast and Pendergrass have shown, the anatomy may be strikingly brought out when the roentgenograms have been made under proper conditions. I agree with Dr. Pancoast's view of the mechanism of swallowing. I believe the larynx does elevate and comes into contact with the pushed-down epiglottis, and that the larynx is in that fashion closed off. In regard to foreign bodies in the upper air passages, you as clinicians are interested. Frequently we find a foreign body that may have been there a long time. A pencil eraser was found months later in the nose, because a roentgenogram was taken after persistent epistaxis. In another case complete stenosis resulted after a dental plate had been in the larynx for seventeen years. Children have carried safety-pins in the lungs for months. One case went along comfortably on tracheotomy, but when the tracheotomy tube could not be removed the child was sent to Dr. Chevalier Jackson, who found the pin and removed it after it had been there for nine months. An open safety-pin is apt to lodge in the upper air passages and may penetrate portions of the larynx. Thumb tacks are apt to penetrate the pharyngeal wall. Fish bones may go almost to the junction of the pharynx and esophagus. Diagnosis of nonopaque foreign bodies in the pharynx cannot be made unless swelling is produced. A fish bone or splinter cannot be eliminated by *x*-ray. New growths and abscesses have been shown in this excellent collection of Dr. Pendergrass, and I can but express my admiration for the presentation.

Dr. Fielding O. Lewis: I think we are all very much indebted to Dr. Pancoast and Dr. Pendergrass for this presentation. The roentgenologists are becoming indispensable as far as laryngologists are concerned. I do not think there is any doubt that the larynx is elevated, as may be demonstrated

in laryngotomy. There is astounding strength of the larynx in its closure. A finger introduced into the larynx in laryngectomy is caught in a contraction sufficiently great to be painful to the finger. In the plates taken after laryngectomy I found much of interest. Previously I thought the buccal voice development was due to the swallowing of air, which was later used; but here we have been shown conclusively that the air chamber is in the upper part of the esophagus and lower part of the larynx. I hope Dr. Pancoast can show what acts as a pseudoglottis. The posterior pillars of the pharynx may be this pseudoglottis, as appeared to be the case in 2 of our patients who were nasopharyngoscoped. Sometimes the manner in which the tissues heal may leave structures which may serve as a pseudoglottis. In the diagnosis and treatment of malignant disease, such observations have been of great value. If they can tell us when the disease is limited, we have hopes of saving the lives of some of these patients by radical surgery and *x*-ray treatments.

Dr. Gabriel Tucker: The bronchoesophagists and roentgenologists have been closely linked together by Dr. Chevalier Jackson. Success in the bronchoscopic clinics depends upon the efficiency of the *x*-ray department, and success in bronchoscopy depends on support from the roentgenologists.

FEBRUARY 18

How Dr. G. Hudson Makuen Treated Stammering

Mrs. Mary S. Steel (by invitation): It is difficult to epitomize Dr. Makuen's work, but the many reprints which are available are each complete articles with technical detail. It is to be regretted that more attention has not been given to nomenclature, especially in the direction of simplification. Dyslabia must be differentiated from pseudolabia. Preventive measures are directed toward three stages of stammering—prodromal, acute and chronic. The time to cure stammering is before it begins. Speech and mental development occur simultaneously. Normal speech is largely automatic. When a child hesitates, it does not know how it happens nor how to stop it. The difficulty suddenly occurs with excitement, increased heart action and general tension. Repetition occurs. This is the prodromal stage, and it is here that the turning point occurs. The child should not be scolded, ridiculed or admonished against stuttering. No references should be made to relatives or ancestors who had improper speech. Mishandling at this stage results in the appearance of acute manifestations. If the individual is tided over the prodromal period, progression is halted and the remedy is easy.

To save the child from the second or third stage, do not permit the hearing of rapid speech. Do things slowly and deliberately and raise the standards of speech for the other members of the family. The child is encouraged in play to repeat speech. There should be plenty of sleep and no exciting influences. Fear of speech leads to the second or acute stage. The treatment of the acute period is similar to that of the first stage, and consists in keeping the child from school and training it in speech mechanism. No reference is ever made to speech defects. Training is best done in a hospital school and away from the brothers and sisters. In the third stage the defect has fully developed, with psychological variations and fear predominant. Fear is a characteristic of all chronic stammerers. It is useless to tell him that his fears are groundless. The one thing uppermost in his mind is that he cannot speak as others do, that he is out of tune and out of control. He is self-conscious, and although he desires to speak, he knows he cannot and the disability becomes more marked.

The predisposing causes are inherited or congenital. The tendency to stammering may be transmitted from the parents. Stammerers appear to be unable to arouse certain auditory images. The exciting causes may be due to peripheral obstruction or to a rapid, excitable environment. In the chronic stage the only salvation is to go back to the beginning. A new type of speech must be developed, as well as a new superstructure. The articulatory mechanism is only for steering; no speech is obtained without proper cerebration (carburetion). The patient is trained to use only the movable parts of the respiratory mechanism. A full breath should never be taken before speaking, but merely the normal quantity as in the military position. Much practice is necessary.

Stammering is generally due to faulty or delayed phonation. Voice culture should be essential. The physical voice produced is not the same for speaking and singing. Speech culture is the more valuable. The voice must be made to take first place in speech, producing phonation rather than articulation. Control is acquired by volitional effort. Many stammerers are profoundly neurotic, but the neurosis is more resultant than causative.

The treatment is therefore approached through the physical rather than the psychical. Many cannot be approached through any other avenue than speech. Hypnotism has been used. Treatment to be effective must be psychophysical. No two stammerers are alike and the treatment is different in each case.

Discussion. Dr. George M. Coates: Some of us will remember that Dr. Makuen impressed on us, fifteen or twenty years ago, the importance of studies on defects of speech. Several times a year he would read papers and present patients, but his was a continuous struggle against the inertia of the profession, and he succeeded in interesting otolaryngologists only to a limited extent. Success in this field requires much time. One cannot practise otolaryngology and also treat defects of speech, for the latter is a specialty in itself. Stammering has been divided into three stages and the proper cure is accomplished before it has begun. Many in the medical profession and individual's family will regard the condition as temporary and firmly believe that the child can be "laughed out of it." Poking fun and mocking of the child by playmates makes matters much worse. The important point is the training of the family and the child's companions rather than the child.

Dr. James A. Babbitt: I think most of us have felt helpless in facing this proposition of stammering. In my class at Yale a certain boy stammered and practically wallowed in his efforts to speak. Twenty-five years later, at a class reunion, the president introduced the old stammerer as the speaker of the evening, and he made a brilliant oratorical impression. A relative of mine by marriage had a harelip, later stammering and infantile paralysis. He landed at Cornell and found there a school which taught him a curious manner of speech. Within a year he spoke extremely well.

Dr. B. Alexander Randall: I ought to be competent to speak on this subject since I occupied the next room to Dr. Makuen for some years. It struck me as curious that, in some of these cases, hesitation is greatest when conversational efforts are made, while the person may orate fairly well when on their feet. Some of my associates were fluent on their feet rather than in general conversation. There are decided differences between those who are victims of this infirmity, and there are many different methods of correction. Some can chant but have difficulty in conversation.

Mrs. M. S. Steel: I want to thank Dr. Coates for his great interest in this work. Ethical work is being done in schools by teachers and some of it is excellent work, but this is work which should be done before the child goes to school, and the medical profession should correct it before school age. Much unethical work still goes on. The man Dr. Babbitt mentioned did a lot of hard work in those twenty-five years. I have seen many cleft-palate cases, but never one that stuttered. In order to correct their speech we must teach them to make sounds they were unable to make with a cleft-palate. The Martin School in Ithaca may be the one to which Dr. Babbitt refers. Unethical schools frequently make use of rhythm. The singer thinks of tune rather than words. I knew a physician who, as a student, studied Dr. Makuen's methods because he had a hesitation in speech, but he thought the thing that helped him most was impersonation (Southern drawl, Scotch, etc.) and then could speak with difficulty. He kept this up until he could speak English well. In reference to Dr. Randall's remarks we should recall that Charles Kingsley could preach brilliantly, but stammered in conversation. Many stammer, but sing well or speak well. It is necessary to phonate, articulate and coördinate with the mind correctly.

Each person's voice should be as low as consistent with free vibration. The case Dr. Babbitt mentions had assumed a falsely low tone which attracted the attention of the patient and aided him in speech.

Experimental Proof that Voice Cannot be Maintained at a Level

Dr. Douglas MacFarlan (by invitation): The criticism that the voice test of hearing is unreliable is justly based on the fact that voice intensity cannot be maintained at a chosen level. In the experimental work of making a constant intensity record for use with the phonograph-audiometer, a list of monosyllables were chosen from the vocabulary of the preschool child. In other terms, the simplest and most familiar words of the language were chosen. This was done with the purpose of reducing to a minimum the factors of intelligence, memory and education. The words were also selected to some degree so as to cover the variety of vowels and consonants, due consideration and elimination being made for words acoustically difficult of interpretation. The records were made at the Victor R. C. A. recording studio by Mr. Erdman, who has been occupied for many years with voice recording and with acoustics of the voice. Great care was taken to maintain what appeared to be a constant voice intensity level. Three records were made; in each, the same series of words were used. It was found that the intensity of the words varied considerably. The words were spoken with different force. In using this type of record in testing the hearing and interpretation of the human voice, it is interesting to note that, when controlled by rheostats, the intensities are dropped to a point near the hearing threshold, it is the consonants that are misinterpreted and not the vowels. In this study the words are merely considered in the crudest light of their total intensity. Pitch analysis and the study of the sequence of the various fundamentals of the vowels and consonants would supplement the above study. Our records show that the interpretability is not due solely to the loudness of the words.

Discussion. Dr. Philip Stout: Are we to understand that a person in normal voice in the same tone varies from 20 to 45?

Dr. MacFarlan: What Dr. Stout brings up is interesting because it seems so remarkable. The man who made these records is well trained and knows acoustically what he is doing. If you heard him you would think

the intensities the same. Acoustically the force of different words are different. Some are explosives. One of the key considerations is that we hear things largely according to loudness of them. We hear chiefly because a thing is loud enough to hear.

Lesions of the Pterygomaxillary Fossa (Report of Three Cases)

Dr. Robert J. Hunter: It was shown that the pharyngomaxillary fossa may be infected by way of the various important structures that are contained in it or surround it. Due to the position of the fossa, abscesses in this region do not tend to point on the surface. Incision should be made early when dealing with abscesses in this area.

Three cases were reported; 1 arising from the ear, 1 from a peritonsillar abscess and 1 from the lymphatic glands. One case, that of a female, aged forty-five years, had been admitted with a diagnosis of pneumonia, after an illness of ten days during which time a peritonsillar abscess had ruptured spontaneously. The glands of the neck later became swollen and the patient became dyspneic and expectorated blood-tinged sputum. Several hemorrhages from the mouth followed, one of 600 cc. occurring after admission. Swelling of the right arm followed, with red streaks following the lymphatic vessels, and the right leg showed general redness and swelling. Direct laryngoscopy revealed long strings of blood clot in the larynx and trachea, the dyspnea disappearing following the removal of the clots. A large black mass in the left tonsillar region pushed the palate to the right. The left tonsil was entirely eroded, and the removal of clots revealed a large bleeding cavity. The common carotid was, therefore, tied and no abnormality of the jugular vein was noted in the exposed area. There was no pus about the great vessels. The patient had no more hemorrhage, the wound remained clean, the throat improved and there was no more dyspnea. Fluctuation developing in the left thigh and the arm. Surgical drainage was made and pus obtained, but later the entire subcutaneous tissues of the arm and leg seemed to break down. The temperature ran a low course throughout and was never of the septic type. Streptococcus hemolyticus, Staphylococcus hemolyticus and Micrococcus catarrhalis were cultured from the leg abscess. The throat cultures were

negative. One month and two days after admission the patient died and autopsy permission was denied.

The second case, that of a colored female, aged forty-five years, had had a discharging right ear for sixteen years. Menstruation had ceased following a gynecological operation eight years before, but since that time she had bled from the ear every month. Five days before admission she awoke with a pain on the right side of the throat and noticed an ulcer in the tonsillar region. Swelling on the right side of the face anterior to the ear followed and became so extreme that chewing became impossible. On examination, a large, nonfluctuating, tender swelling was found in the parotid region, with swelling of the parotid orifice, but no pus was expressed on pressure. The external auditory canal was full of pus and was nearly occluded by a swelling half-way toward the drum; but no pain, tenderness or swelling was noted over the mastoid. The anterior pillar of the tonsil was red; the tonsils were not involved. The posterior pillar and adjacent side of the uvula were slightly edematous. The palpating finger could detect a fulness in the right lateral wall of the pharynx. On the day after admission the patient suddenly became stuporous and a preoperative blood sugar was reported as 402 mg. with a blood urea of 40. Operation was postponed and the patient transferred to the metabolic service. The following day the mental condition was more clear but the swelling over the parotid became more marked and a symmetrical, bilateral, submental swelling appeared. The medical service was of the opinion that the blood-sugar and blood-urea nitrogen were not sufficiently high to explain the comatose condition and the neurosurgical consultant was brought in. Finding the pupils contracted and fixed to light, and reflexes lost in all extremities, with flaccidity of the left arm and leg, a diagnosis of left hemiparesis, probably due to an extradural abscess in the middle fossa rupturing through the foramen ovale into the neck, was made. The best avenue of attack appeared to be through a radical mastoid operation and this was done under local anesthesia. The mastoid process was not markedly involved, but the antrum and middle ear were filled with cholesteatoma, polyps and pus. The dura was normal. The middle ear was thoroughly cleansed and necrotic bone curetted in the floor of the tympanum. A great amount of foul pus welled up from the neck. The patient expired on the table. Spinal-fluid examination postmortem was negative. Autopsy revealed a large

abscess in the pharyngomaxillary fossa. A probe was passed through the carotid artery and jugular vein with ease. There was no evidence of thrombosis or extradural abscess. The meninges were normal. The right half of the brain was markedly anemic.

The third case, that of a boy, aged fourteen years, developed a sore throat some years after tonsillectomy. This subsided, but later the patient noted a severe pain back of the left jaw, inability to open his mouth freely and pain in the ear. Several days later the drum became mildly suffused and light pink in color and the jaws could only be opened with great difficulty. There was edema at the base of the tonsillar fossa. Back of the posterior pillar in the left lateral wall of the pharynx was a sausage-shaped swelling with some fluctuation, leading to a diagnosis of abscess in the lateral wall of the pharynx. An incision into this mass did not reveal pus. The next day the abscess opened at a higher plane back of the palate. Recovery was uninterrupted. This was apparently a case of pharyngomaxillary-fossa abscess due to the breaking down of an infected lymphatic gland.

Discussion. Dr. Temple Fay: I was much interested in the case I was called to see. Dr. Petty felt that the profound coma could not be due to diabetes *per se*. The patient had a clear-cut lesion involving the 5th, 6th and 7th nerves on the right and, therefore, outside the dura. The lesion was a left-handed paresis. The Babinski was bilateral. This escaped from the neurosurgical standpoint because of cerebral edema, and we felt there was not only an extradural lesion but that it was also extracranial. These cases are very interesting and present a most interesting symptom-complex. Some of them do not produce pus, and one does not always find a mass of pus above. The nonproductive type may involve only the 5th and 6th nerves. The plucking of a hair in the trigeminal zone produces excruciating pain. We also get a prompt anisocoria, and this anisocoria is diagnostic. Pain in the carotid may be reflex even from as low as the gall-bladder. The important point in Dr. Hunter's paper is, "What caused the stupor." In 70 per cent of cases the longitudinal sinus turns to the right and the left lateral sinus drains only the veins of Galen. The predominance of the large sinus is to the right. Desperate cases are frequently associated with right-sided pathology. As shown at autopsy, this patient had a foramen hernia. Bilateral cerebral edema was present. Occlusion on the right side is more important than on the left. I believe this woman had obstruction interfering with drainage and producing cerebral edema. I do not know whether the patient died of the anesthetic or obstruction. The mechanism of the right jugular is the interesting point to follow here and this case interestingly illustrates a right-sided lesion.

Dr. O. V. Batson: The pterygo- or pharyngomaxillary fossa is usually not clear in our minds. Two well-defined fossæ or collections of areolar

tissue exist in these areas. I believe that anything in front of the vessels belongs to the pterygo- or pharyngomaxillary fossa. The tonsil is at the upper pole of the line of bloodvessels running up the neck. If anterior, the location of pointing is beneath the mandible; if posterior, the mediastinum. It is always possible to open through the mouth if the infection has not progressed too far. Any bloodvessel crossing bone has its thin side toward the bone. Any condition causing erosion of the temporal bone comes down where the bloodvessel is thinnest. Many skulls have bone missing in the apex of the petrous portion of the temporal bone. Drainage above the palate was probably traveling in the prevertebral fascia.

Dr. B. Alexander Randall: In my study of the temporal bone (1000 or more preparations) I found a right-sided preponderance in size of the lateral sinus in 60 per cent.

Dr. David Husik: In this case it was decided that intracranial interference and metabolic management were not vital, so that the otolaryngologist was forced to assume responsibility. The external canal was filled with polyps. It was decided to do the mastoid operation under general anesthesia. I have thought that it might have been better to tie off the jugular before proceeding.

Dr. Karl M. Houser: We frequently tie the internal jugular vein, and in several reported cases both jugular veins were tied. We have not feared cerebral edema in the cases tied on one side alone.

Dr. Temple Fay: Given a block in the venous system and given time, compensation occurs. Compensatory circulation is established before operation and for that reason no damage is noted. If the vein is tied suddenly, then pressure and edema occur. The cortical supply is drained often to the right and the cortical supply is important for consciousness.

Dr. O. V. Batson: The French describe four types of torculæ, and with arrows show the direction of bloodflow. Osteology books show this variation. What happens when you ligate the jugulars? Skulls differ enormously. In acute jugular obstruction trouble will ensue unless there is sufficient extrajugular drainage. The posterior jugular vein, described by the French but not insisted upon by the Germans, frequently drains the mastoid area.

Dr. B. H. Shuster: Dr. Hunter's paper brushes up our anatomy and physiology, and Dr. Batson's discussion has added a great deal. In normal anatomical necks we find textbook structures, but when swelling occurs the anatomical differentiation is frequently lost. Dr. Dandy describes consciousness situated in the left lobe. We need Dr. Batson with us to correlate these things.

Dr. Robert Hunter: This brain was not tremendously edematous; it was a microscopic rather than a macroscopic finding. The brain on the side of the lesion was anemic; the opposite side had congested vessels. On microscopic examination, on the affected side, the cells showed poor staining, with a tendency to the so-called "ischemia cell disease" and showed tremendous glial increase. There was considerable edema on both sides, not more on one side than on the other. Blocking of the carotid may have occurred since the brain was anemic on that side. The onset was sudden and there was not tremendous edema of the brain.

MARCH 18

Postoperative Treatment by Radium in Radical Mastoidectomy

Dr. Joseph C. Keeler: Enough work has been done to demonstrate that the use of radium in ear work is something more than a possibility, but more clinical evidence must be available to extend its usefulness and establish its actual worth in certain otologieal lesions. Fifty cases in which radium was employed as an adjunct to the postoperative care were presented. In otology the usefulness of radium is almost entirely outside the field of malignancy, the field in which it is most commonly employed and most closely associated. The cases presented had had radium applied during the healing of mastoidectomy wounds, and in all cases radium was used only after surgery. Radium should never be regarded as a substitute for proper surgical measures. Only after necrotic bone and damaged tissue have been removed can efficient work be performed by radium. The labor it performs is regenerative—not destructive. Its curative properties depend upon its power to build up damaged cell-structure.

Coöperation between the otologist and roentgenologist is essential to the successful use of radium in otology. While the action of radium is lethal in large dosage, in moderate quantities it is stimulative. Occasionally we encounter conditions which are beyond the limits of even the most skilfully performed surgery, where life may be dependent upon the preservation of a blood-vessel threatened by advancing necrosis which the surgeon is powerless to stay, or paralysis may be imminent by reason of the encroaching disease upon vital nerve trunks. A circumscribed suppurative labyrinthitis, as a necrotic process, may erode through the petrous pyramid, exposing the internal carotid artery. Here radical surgery must be first in order, but what this cannot fully accomplish may be brought to a successful termination by the judicious use of radium. Where discharge persists from tympanum and Eustachian tube, or dermatization is delayed because the formation of granulations is slow or wholly lacking, it has proved of the greatest possible assistance.

As a routine the radium emanation containers were filtered with 1 cm. of wood and 1 mm. of lead, except when the container was

a 12.5 mg. platinum needles inserted directly into the middle-ear cavity. Most of the cases reported had previously undergone radical mastoidectomy, and a number of them had had several so-called "mastoid operations" before coming under observation. A few were cases in which radium had been used because surgery for some reason was contraindicated. In circumscribed suppurative labyrinthitis, where opening of the labyrinth may very well usher in a generalized infection, with meningitis and death, or where the surgeon is convinced that he will induce facial paralysis if his work is extended further, the use of radium before further intervention should always be considered. It is always possible to operate later if radium proves ineffectual. Where fibrous growths persisted in the region of the oval window, or there were polypoid formations following radical mastoidectomy in fistulous openings in and about the cochlea, two or three sessions, totaling from three to six hours, with 50 or 100 mg. applicators, proved adequate to remove the new growth and permit dermatization. In one patient, a large fibroma clogged the meatus, but was rapidly removed after radium treatment; in another, there was persistent return of the granulations in the region of the oval window, as well as persistence of the fistulæ in the same location.

Cost is no longer an objection, since every large center has a supply of radium, available either in the form of applicators containing the element, or maintains a radium emanation plant, by means of which the cost and flexibility of this therapeutic aid are regulated.

Discussion. Dr. James A. Babbitt: Does the radium have a direct effect in stopping the latent discharge which follows radical surgery?

Dr. Philip Stout: How many failures were there? What was the percentage of failures? Were any bad effects noted in occasional cases in this series?

Dr. Joseph C. Keeler: In some cases with persistent discharge excellent results followed one or two applications and they became dry. We did not obtain 100 per cent success, for this medium is merely an adjuvant. In the great majority of cases it does prove very effectual.

The Usual Course and Relations of the Nervus Facialis and the Practically Important Variations

Dr. O. V. Batson: The portion of the facial nerve within the temporal bone is alone considered. It is divided into five topo-

graphical portions: (1) meatal, (2) geniculate, (3) tympanolabyrinthic, (4) arcuate and (5) mastoid. The arcuate portion curves over the oval window, becoming the more or less vertical mastoid portion. The meatal portion is of interest (chiefly to the neuroötologist) because of its relation to the infratentorial arachnoid space. The geniculate portion is exposed to the dura of the middle cranial fossa in childhood and sometimes throughout life. The canal of the tympanolabyrinthic portion, lying in the wall common to the internal and middle ear, is perforated by small nerve foramina and is the site of dehiscences. The arcuate portion, with the lateral semicircular canal, lie in a dense mass of bone which forms one boundary of the aditus ad antrum. The other two walls of this triangular constriction are the posterosuperior wall of the external canal and the tegment tympani.

The mastoid portion begins at the level of the eminentia pyramidalis, which houses the m. stapedius. Between the cavity in the eminentia and the facial canal there is a foramen for the n. stapedius and sometimes bone deficiencies. These openings, and that for the chorda tympani, lower down, are potential routes of communication between the cavum tympani and the mastoid portion of the nerve.

A landmark, undescribed, for the mastoid portion of the facial canal is the tympanomastoid suture. During the course of an operation this may be located with a probe. The facial nerve lies medial to this suture at a depth which depends upon the lateral thickness of the mastoid process.

The distance between the suprameatal spine (Henle) and the sigmoid sinus determines the ease with which the antrum tympanicum can be approached. When this distance is short, more space is obtainable by removing the posterior meatal wall. The other wall, about the sigmoid sinus, must be avoided because of the sinus and because of the mastoid portion of the facial nerve.

Discussion. Dr. Edward B. Gleason: Dr. Batson is the keenest observer I have ever known. He has given us a simple way of testing the depth of the mastoid process. The facial nerve passes through the internal auditory meatus anterior to the auditory nerve. At the bottom of the meatus the facial enters the facial canal, whose course it follows throughout. At first the canal is directed horizontally outward, then bends abruptly backward, passes above the fenestra ovalis and turns downward, behind the pyramid, in the posterior wall of the tympanic cavity, to end at the stylomastoid foramen. There are other means of getting the position of

the stylomastoid foramen. If in a basal case we run the probe along the digastric groove posteriorly, the facial nerve will be reached. The "ridge pole of the digastric fossa," always at its extremity, is over the stylomastoid foramen. With these matters in mind there is no great difficulty in locating the stylomastoid foramen and we know that the course is in a straight line from that point to the posterior border. Dehiscences are not uncommon and persist in adult life. There is great thinness of bone over the horizontal portion of the nerve in many skulls. The facial nerve has always been a bugbear. What has been said tonight makes it a simple matter to outline the facial nerve and avoid it.

Early Intrinsic Cancer of the Larynx—Diagnosis and Treatment

Dr. Gabriel Tucker: At the risk of repeating the commonplace, I should like to emphasize the following with reference to diagnosis and treatment of early intrinsic cancer of the larynx.

First, chronic hoarseness is an early and constant symptom in practically every case of intrinsic cancer of the larynx.

Second, anterior intrinsic cancer of the larynx, lesion limited to the anterior two-thirds of the cords, metastasizes late, owing to the peculiar lymphatic arrangement of the larynx. Cancer in this locality may be said to remain "early" until evidence of metastasis or extension occurs. It is frequently six months to two years after the development of hoarseness before evidence of metastasis appears.

Third, the operative removal by laryngofissure of the growth while the cancer is still early will cure from 75 to 80 per cent of the patients in properly selected cases, thereby saving the patient's larynx as well as his life. If all cases operated upon are carefully followed and those in which recurrence takes place are treated by laryngectomy, at least 90 per cent of these cases will be saved by laryngectomy.

These facts have been stated repeatedly to the profession but apparently are not fully appreciated, because those of us who are doing laryngeal surgery are constantly seeing intrinsic cancer of the larynx far advanced, where the diagnosis could have been made early if sufficient attention had been paid to the symptom of chronic hoarseness and proper diagnostic methods employed.

Any patient over eighteen years of age with chronic hoarseness should be suspected of having cancer of the larynx until careful study proves it otherwise.

The routine of study in these cases has been stated many times: The first procedure is careful mirror examination and study of the larynx; general studies, to exclude tuberculosis, lues and all other conditions that might produce a lesion of the larynx; roentgen-ray examination of the neck and chest and examination of the sputum to exclude tuberculosis should be made; blood Wassermann and other necessary examinations to exclude syphilis should be included.

In making the differential diagnosis prior to operative removal of a lesion from the larynx, the final step is direct examination and biopsy. This should be done in every case, and in my experience I have seen no bad results from this procedure. I have never regretted doing a biopsy as a means of differential diagnosis, but I have regretted very much my inability to obtain permission for a biopsy until the lesion was far advanced. Early biopsy will frequently reveal a "precancerous condition." Operation done at this stage of development of the lesion will have a much higher percentage of cure than if it is postponed until the lesion is so well developed that its nature is perfectly evident on mirror examination.

I should like to report some observations on a series of 25 cases of early intrinsic cancer of the larynx on which I operated at the Bronchoscopic Clinics of the University of Pennsylvania, at the Graduate and University Hospitals. There were 22 males and 3 females. The oldest male was seventy years, and the youngest male forty-eight years of age. The oldest female was fifty-two years of age and the youngest twenty-six. The first 15 cases have shown no evidence of recurrence, with duration of cure in each case as follows: Case number one, six years and nine months. Case number two, six years. Case number three, four years and seven months. Case number four, four years and seven months. Case number five, four years and five months. Case number six, four years and two months. Case number seven, four years. Case number eight, three years and six months. Case number nine, three years and one month. Case number ten, three years. Case number eleven, three years. Case number twelve, three years. Case number thirteen, two years and eleven months. Case number fourteen, two years and eight months. Case number fifteen, two years and six months. Case number sixteen, after one year and ten months showed some thickening at the site of the original lesion on the left cord. He is at the present

time under suspicion and if recurrence is present will be advised to have a laryngectomy, which will still remove the lesion as there is no evidence of metastasis outside the larynx. Case number seventeen shows no recurrence after one year and ten months. Case number eighteen showed a recurrence after nine months, laryngectomy was not done, radiation was used, he developed a perichondritis and died three months later. Case number nineteen, no recurrence in one year and eleven months. Case number twenty, no recurrence in one year and nine months. Case number twenty-one, no recurrence in one year and eight months. Case number twenty-two, no recurrence, in one year and five months. Case number twenty-three, shows no recurrence at the end of eleven months. Case number twenty-four, showed evidence of recurrence after one year and six months. A second operation, thyrochondrotomy, was done with no recurrence at the end of eight months. If there is evidence of further recurrence in this case a total laryngectomy will be done. Case number twenty-five has no recurrence at the end of four months.

In the first 22 cases there has been 1 recurrence after nine months. Laryngectomy was not done because the patient's general condition would not permit. Radiation was used and the patient died at the end of three months.

A second in the series had a recurrence that was removed by thyrochondrotomy without further recurrence. Laryngectomy can still be done if the growth recurs, as there is no evidence of metastasis.

A third case is under suspicion of further growth of cancer after one year and ten months. Laryngectomy can still be done in this case, as there is no evidence of metastasis.

The percentage of lasting cures to date in this series is 87.5 per cent, with a probability that the final results will remain around 80 per cent, as has been reported previously, when improved methods of laryngofissure had been used in properly selected cases.

The technique used in these cases is that devised by Dr. Chevalier Jackson and Sir St. Clair Thomson, with slight modification as to the closing of the wound, etc. Intratracheal ether was used in 12 of the 26 operations. Skin infiltration with 0.5 per cent novocaine and intralaryngeal application of 10 per cent cocaine was used in 14 operations. The period of convalescence averaged about three weeks. The longest was three months, in a patient in which a perichondritis

developed. The patient was a minister who had an arrested case of pulmonary tuberculosis with a purulent tracheobronchitis. The purulent tracheobronchitis was probably a factor in the development of the perichondritis. He is now well, has a good voice and able to follow his profession.

There was no operative mortality and every case developed a useful voice.

Conclusions. 1. *Early* intrinsic cancer of the larynx will be recognized while it is still *early* when the profession realizes fully the possible significance of chronic hoarseness. Biopsy should be the final step in the diagnostic study.

2. Early anterior intrinsic cancer of the larynx is amenable to cure by laryngofissure, which saves the patient's larynx as well as his life with no operative mortality. The anterior intrinsic groups includes a large percentage of all cases of intrinsic cancer of the larynx.

3. Laryngectomy will cure a large percentage of the recurrences, after laryngofissure, if the cases are properly followed. Partial laryngectomy with postoperative radiation has given very satisfactory results where there was a contraindication in the patient's general condition to laryngectomy in several cases not included in this series.

Discussion. Dr. Fielding O. Lewis: I would like to emphasize one of the points which Dr. Tucker mentioned, *i. e.*, diagnosis. The wonderful pictures which Dr. Tucker has shown are apt to lead you to think that diagnosis is easy, but this is not always the case. Dr. Clerf and I have removed several specimens from one man and all the specimens have been reported to be inflammatory tissue. In spite of these reports we still feel that it is malignant. Frequently patients are referred with a diagnosis of cancer, but when the tissue is examined it is found that the preparation is poor and no diagnosis is possible. This points out the need of a good pathologist with experience and exceptional technique. Dr. Tucker has shown you the two techniques described in the textbooks. I have used electrocoagulation in a number of cases, with no bleeding; but care has to be observed in its application here. We must be careful of our use of the term "cure" in cancer. I have seen cases with recurrence after twelve or fourteen years.

Dr. Gabriel Tucker: Possibly I did not say enough about the use of the mirror. The biopsy is done at the final examination. Where there is questionable diagnosis we always refer the tissue to a number of pathologists. We use the term "lasting cure"—meaning a cure for so many years—a term borrowed from Sir St. Clair Thomson. This does not mean that the patient may not develop cancer on the other side or somewhere else. Thus the "cure" may last four, six or eight years.

APRIL 15

A Modified Attic Drainage in Chronic Suppurative Otitis Media. Preliminary Report

Dr. James A. Babbitt: Twenty-three cases, some bilateral, in all, 29 individual ears, operated upon during the past three years, were presented. A majority present cured "dry" ears at this time. All the cases were improved. The hearing level has been raised and the complications were confined to 1 case (a rotary nystagmus and vertigo of three days' duration), which might belong to the rather severe mastoid exenteration on the opposite side, performed at the same time by an associate.

In chronic diseases of the middle ear the attic space becomes badly damaged. It is conceded that at least the antrum of the mastoid shares the involvement. The squamous-cell areas—promontory, ossicles and tympanic membrane—afford special opportunity for fibrous change. The whole area may be semiobliterated by thickening, sclerosis and adhesions, so that drainage downward from the iter ad antrum remains but a narrow, tortuous channel defying simple treatment. It seemed possible, if this upper posterior quadrant could be converted into an open rather-rectangular drainage, even at further sacrifice of tympanic membrane, but preservation of the annulus, that the trouble might spontaneously cease, that even diseased ossicles freed from surrounding pressure might regenerate or cover in their surface sufficiently to become innocuous, and even after this somewhat traumatic disturbance carry audition value.

The age range of the cases chosen was from four to seventy-four years; duration of discharge from six weeks to fifteen years, though this time period rather strangely seemed to bear little relation to success or speed in recovery. Three important preliminary conditions are important. The mastoid, except for its antrum, should be in a quiescent state; contributing factors, such as adenoids, infection in the posterior ethmoids and active infection in the auricular canal or glands about the ear, should be eliminated; only improvement should be forecast to the patient, as a second or third procedure, might be necessary.

The operation requires but a brief period under nitrous oxide and oxygen; has little pain in sequence and repetition in finally

controlling drainage should not be contraindicated. The procedure is to work from the posterior inferior quadrant. If perforation is anterior, lateral or inaccessibly high, make a fresh incision and continue up under the portion of tense and full depth of flaccid membrane in the posterior superior quadrant, reaming this space out with blunt right-angled probe. After cleaning this space thoroughly, bite off portions of the overhanging membrane to secure free drainage. Push aside if necessary, but avoid removal of any portions of malleus or incus. If the former protrudes down into the lower enlarged opening, later healing will render this stationary and perhaps still somewhat effective in sound transmission. The final step is to insert angular curettes reaching to the neighborhood of the iter ad antrum and curette away any blocking granulations. It would seem possible for this to disturb the facial nerve as it passes down beneath the passage from attic to antrum, but this accident has not so far occurred, nor has any nerve irritation been noted.

The after-treatment has been more or less empirical. The patient has been kept in bed about three days. At the time of operation a strip of gauze packing is inserted, dipped in mercurochrome or metaphen solution. This is allowed to remain the first night. The following day the ear is irrigated by a bulb syringe with normal saline solution, the canal dried with cotton and a few drops of 2 per cent phenol in glycerin instilled into the canal. This is irrigated at first every three hours and after the first thirty-six hours reduced to three times daily. In four or five days the discharge lessens, active irrigation is stopped, the ear is wiped with a cotton swab and touched with strong metaphen solution. It is quite possible that all this irrigation is superfluous, but conditions are not similar to early acute catarrhal or acute suppurative involvement of this area. The single important point in the immediately convalescent period is to maintain a sure drainage.

Discussion. Dr. Oscar V. Batson: Dr. Babbitt performed his operation on several cadavers on which the tegmen tympani had been removed, revealing the malleus and incus. I was looking down from above while Dr. Babbitt was carrying out his technique below. As he worked he described the location of the instrument and invariably the instrument was in the location he described. Limiting the opening automatically prevents interference with these ossicles. The question might arise as to injury to the stapes and the oval window. Judging by the density of the

attachments the foot-plate portions of the stapes would give way before the foot-plate was dislodged from the oval window.

Dr. George M. Coates: Dr. Babbitt has well said that chronic suppurative diseases of the ear are the bane of otolaryngologists. Any method which will clear up some of these is valuable. We are between the extremes of radicalism and conservatism. When there is little mastoid infection we should employ intratympanic methods as far as possible. Even ossiculectomy may be contraindicated because of deterioration of hearing. Dr. Babbitt's plan sounds simple and feasible. We all know that if you can get free drainage from the cavity, half of the battle is won. I see no reason why, if the discharge is limited, the diminution of deterioration of hearing should not follow. By removing granulations and breaking up adhesions we should get improvement rather than diminution.

Dr. Edward B. Gleason: It is a well-known fact that many chronic otorrheas due to attic suppuration get well if cleanliness is observed. The incus is frequently separated from the stapes and the stapes lies free in its niche. Cholesteatoma frequently when removed result in abrupt cure. Even the removal of a polyp may result in cure. In using an instrument devised to remove polyps from the antrum I once removed the stapes and Dr. Stout, about the same time, had the same unfortunate experience. Perilymph discharged for some days with some vertigo and then the condition cleared up. As far as necrosis is concerned, the superficial necrosis will take care of itself if cleanliness is observed. In most of these cases sclerosis of the mastoid process has occurred, with diminution in the size of the antrum. Often a fistula extends from the antrum into the necrosed mastoid and is the source of the discharge. Such a method would prove ineffectual in these cases. Stacke's operation is occasionally successful, but not as uniformly successful as the radical mastoid operation. I must say that in Philadelphia all of us are getting results from the modified radical operation in which the contents of the tympanum are preserved.

Dr. J. C. Donnelly: I saw a child at the Children's Hospital with a history of three years' suppuration, and we were anxious to do a mastoid operation. Later I saw the child after Dr. Babbitt had used this technique and the results were excellent. At the University I saw quite a number of cases similarly treated and the results were uniformly satisfactory. I am certain that this method secures drainage.

Dr. S. D. Weeder: I am interested in this operation and believe it will do much in the successful handling of these cases. I would like to know what type of operation Dr. Babbitt would advise in a case of pure attic suppuration with a punched-out perforation? Should one go in from below or from above?

Dr. F. G. Pressman: I saw a third of Dr. Babbitt's cases. In the case in which nystagmus occurred, following operation, I had uncovered the lateral semicircular canal. Next day nystagmus occurred. The postauricular wound was opened and the area cleaned up. If the nystagmus had been due to interference on the opposite side, a distructive labyrinthitis would have occurred and this would not have cleared up. We are forced to the conclusion that the nystagmus was due to traumatic labyrinthitis

occurring on the mastoid-operated side. The success of this operation is due not only to the drainage. Very early in the course of convalescence we can see with the otoscope islands of epithelium growing over a fibrous-tissue base. We not only secure drainage after the operation, but induce epithelization. The cases do not drain after operation, but remain dry. One case that Dr. Babbitt considers a failure was due to mucoid secretion that entered from the Eustachian tube.

Dr. James A. Babbitt: Dr. Butler asked me why we did not remove the ossicles. Shock is obtained when ossicles are removed. This other procedure takes only five to ten minutes. By leaving the ossicles we may get attachment to the drum and make some audition possible. As to the question of leaving infection behind (we assume that mastoid infection has become quiescent)—by opening up the antrum, healing occurs from above downward and we do not presume blockage from below. One case had attic suppuration and I deliberately made an opening below and cleared up the condition. So in these cases Dr. Weeder mentions I go in low down.

Malignant Conditions of the Laryngopharynx

Dr. Henry B. Orton (by invitation): Drawbacks to operations on the laryngopharynx may be classified into three groups: (1) The uncertainty of cure, which is the most important; (2) the danger after the operation as well as during the operation, and (3) the mutilation and disability that may be left by an operation otherwise perfectly successful.

Intrinsic cancer is being well attacked by both laryngofissure and laryngectomy, but something can be done for patients who are suffering from so-called extrinsic cancer of the larynx. Better terms are epilaryngeal cancer, a growth originating on the epiglottis, aryepiglottic folds, pyriform sinus or lateral wall of the pharynx, and hypopharyngeal cancer occurring in the lower part of the pharynx.

Lateral transthyroid pharyngotomy impressed me as being an ideal procedure by which to attack malignant conditions of the pharynx. I have had occasion to perform this operation on 9 patients, with an operative mortality of 2, or 22.3 per cent, both of the patients dying within thirty-six hours of septic pneumonia. Three patients had a recurrence in the glands six months, and 1 patient nine months, following operation; 1 lived for one and a half years, and 2 are living at this writing, 1 patient now for over one year and the other for eight months.

Microscopically we are concerned only with the squamous-cell epithelioma. Mr. Trotter's classification shows the hypopharyn-

geal group, which involves the cricoid region and the posterior pharyngeal wall, and which is practically limited to women. The group known as the epilaryngeal, which involves the arytenoids, aryepiglottic folds, epiglottis, pyriform sinus and possibly the base of the tongue, is almost entirely limited to men. In the order named, a growth involving the epiglottis, aryepiglottic folds, lateral wall of the pharynx, postcricoid and pyriform sinus gives the best prognosis. If the growth has had its origin from the lateral wall of the larynx, it soon involves the pyriform sinus and thyroid cartilage, and nothing short of complete laryngectomy, with a partial or entire cervical esophagus, will be of any use. This group is the most discouraging one for any operative procedure, yet if the condition is seen early enough a few additional years of life may result from the operation.

After a preliminary tracheotomy the incision for ordinary transthyroid pharyngotomy would be along the anterior border of the sternomastoid muscle, reflecting anteriorly and posteriorly the muscles of the larynx. But as most of these patients, by the time they are examined, have enlarged glands of the neck, from either infection or lymphatic extension, it becomes necessary to perform a resection, and the former incision is inadequate. An additional incision is then made, more or less at right angles to the original one, running from the angle of the jaw downward, forward and upward to the symphysis.

These flaps and the platysma are turned back; the superficial fascia, the submaxillary gland and the deep fascia are likewise removed upward and backward as far as the spinal accessory nerve and posterior triangle. This step exposes the deep vessels of the neck and the muscles surrounding the larynx. All the vessels that come off anteriorly from the great vessels are ligated and cut. The internal jugular is ligated and resected. The external carotid may or may not be ligated. The sternomastoid may or may not be removed, depending on the glandular involvement.

The next step is to suture the sternomastoid to the prevertebral fascia; this covers the great vessels and thus protects them from infection when the pharynx is opened. The area is drained by a counter opening posteriorly.

The following muscles are then reflected from the hyoid bone and thyroid cartilage: the hypoglossal, thyrohyoid, sternohyoid, sternothyroid, stylopharyngeus and the inferior and middle con-

strictors of the pharynx. One is then ready to remove, by means of heavy scissors, the exposed portions of the hyoid bone and the greater portion of the thyroid cartilage. This permits palpation of the growth through the pharyngeal aponeurosis and mucous membrane, and enables one to decide where to make the opening into the pharynx so as to be well around the malignant area; or if the growth is too large to remove, one may back out, so to speak, and not proceed further. The pharynx is then opened. Before operation one has a fair idea of how much tissue it will be necessary to remove, and the flap is made sufficiently large to fill up the defect. A feeding tube is inserted into the esophagus. Later, a plastic operation is performed to close the neck.

Discussion. Dr. Fielding O. Lewis: I have greatly enjoyed Dr. Orton's splendid presentation of this important subject. Dr. Orton has made a plea for early diagnosis. Dr. Jackson frequently has pointed out that many cases of chronic hoarseness, if investigated, would reveal early stages and many lives would be saved. The larynx was frequently not visualized for ten to twelve months after onset in looking over our records. When limited to the vocal cords, the results in cases in which laryngofissure is suitable show 70 to 80 per cent cured. In extrinsic forms of cancer the patient may have few symptoms until the disease is far advanced. They come to us far too late and are usually hopeless cases. Out of 150 cases at the Philadelphia General Hospital, all of which were extrinsic, less than 1 per cent were suitable for treatment. *X*-ray and radium were mentioned by Dr. Orton. We have had no good results with radium and *x*-ray alone, but frequently had good results with surgery, radium, *x*-ray and electrocoagulation. When extrinsic cases were formerly seen the patient was palliatively treated and death was awaited. Now we are saving a few of these cases. Dr. Orton showed you some of these "hopeless" cases still enjoying life after some of these extensive operations.

Dr. Gabriel Tucker: I appreciate these casts Dr. Orton has shown and we would all do well to study them.

Joint Meeting, Sections on Otology and Laryngology; Public Health and Industrial Medicine

MAY 20

Audiometric Findings and Clinical Records in Philadelphia Public Schools

Dr. Walter S. Cornell, Dr. Marie Currie Frey, Miss Roberta Laird: In instituting audiometric examinations in public school

work it was found that considerable data rapidly accumulated which was an aid in the solution of numerous teaching problems, as well as having a direct bearing on school health problems. Many school children who had been listless and deficient in their school work were found to have acoustic loss. The acoustic loss also had a fairly direct relation with certain types of behavior. To a certain extent, knowing the audiometer record, it was possible to predict deficiencies in the scholastic record.

The technical difficulties that required solution were outlined. A portable instrument is essential. The operator must have experience not only in handling the instrument but likewise in handling children of varying ages in order to obtain complete coöperation. No less important is the coöperation of school officials and parents. The medical profession should encourage this work and see that the cases showing definite hearing loss are followed by the general practitioner and by the otologist.

Early detection of otitis media and remedial defects was made possible by these routine examinations. Where the hearing loss was too great for instruction by the usual means, the child was sent to appropriate classes for instruction in lip-reading. There is always the possibility that ear infections may develop soon after an examination and when sudden scholastic deficiency appears a reëxamination is in order.

It would be preferable to examine every child on admission to the public schools in order that defects come early to the attention of the otologist. We cannot, as a rule, reach the child of preschool age, but there is little excuse for missing acoustic losses once the child is in school and under direct supervision of the school health authorities. It is hoped that with the accumulation of further data in various centers much valuable data will enable the physician to deal earlier and more effectively with the school child with deficient hearing.

Noise in Industry, Its Effect on the Hearing and on General Health

Dr. Henry Field Smyth: Although it has been long recognized that extremely loud explosive noises may be the cause of sudden deafness, often due to perforation of the ear drum, found at times in gunners and bellringers, it seemed desirable to consider deafness

as an occupational condition rather than due to explosive trauma. Trunkmakers, riveters and boilermakers usually become deaf. Most middle-aged persons living and working in big cities are slightly deaf, due to the roar and rattle of traffic, motor busses, taxis, engines, sirens, telephones and typewriters. This would indicate that it is not only the extremes of noise that can harm.

It has been observed clinically that high grades of impairment of hearing are more common among those metalworkers whose day labor confines them to subjection to intense sounds made up largely of overtones of high pitch than among workers in noises of low pitch. Impairment has been found in 50 per cent of smiths and machinists examined, and in 35 per cent of coppersmiths, while normal hearing is found in only a little over 9 per cent of boilermakers. Out of 31 boilermakers in one factory there was not one with normal hearing. Workers in a noisy atmosphere develop a true nerve lesion, and increasingly so if the noise is in small closed spaces, as in boilers. Observations on railroad employees show deafness among engine drivers and stokers increases with age and years of service. Deafness has been reported among spinners, weavers and beetlers. The impairment of hearing in all these cases has a definite relationship to the duration and the intensive character of the operative sound. Subjective noises were an almost unvarying accompaniment of the impairment of hearing, but dizziness or vertigo was present only in the more advanced cases where the upper tone limit in hearing was notably decreased. Dizziness is also more likely to be an accompanying symptom in mill and factory operatives where the noise of the machinery is accompanied with a sensible vibration.

The affection of hearing incident to exposure to loud sounds of high pitch is usually thought to be one of the internal ear solely, but Holt thinks that changes in the middle ear and sound-transmitting apparatus are mainly the cause of the impairment. Gradinego distinguishes three types: labyrinthitis without middle ear impairment, chronic catarrhal otitis media with labyrinthitis, and chronic advanced middle-ear inflammation with implication of the labyrinth and vertigo; while Ropke is of the opinion that in the majority of cases both the middle ear and the labyrinth are involved.

Noise can impair hearing by its intensity as well as by its duration. It is transmitted not only through the air, but also through the wall

and floors of workrooms. A single strong sound expression is especially injurious if it occurs suddenly and unexpectedly. The noise injures the most sensitive and most vital parts of the organ of hearing, namely, the auditory nerve endings in the cochlea. Not every human ear is injured by loud continual industrial noise. The greater the power of resistance of the organism, and the healthier the organ of hearing, the less harmful is the effect of noise. Impairment of hearing caused by continuous moderate occupational noise develops gradually and is at first hardly noticed. Examination by an otologist often reveals impairment of hearing in persons not aware of their injury. In the case of impaired hearing there is first impaired preception of high tones; but after prolonged exposure the degenerative process extends to other auditory cells which serve for the perception of lower tones.

Stopping of the ears with plugs of cotton, or of flax fiber, is not sufficient protection against noise in workrooms. Protection should consist of compact plugs of gauze saturated with petroleum or wax. If the floor vibrates, straw, cork or rubber soles or felt shoes may be helpful; for kneeling, felt mats may be used. Apprentices coming from families with hereditary ear troubles should not work in noisy factories. When beginning work in a noisy plant every employee should be examined by an otologist and the examination should be repeated at suitable intervals. Endeavors that strive to limit industrial noise should be promoted and supported. They include the proper distribution of machines and suitable contrivances for the suppression of noise. Relaxation in quiet surroundings when away from work is the most helpful means of preventing ear troubles.

Discussion. Dr. Douglas MacFarlan: As far as noise goes, otologists as well as workers in industrial medicine will be driven by the populace to deal with this question. Secondarily, it is of interest to neurologists. The survey made in New York was a very complete study. Industrial deafness and its damages have been recognized by the Germans, who employ deaf folks in industries which make people deaf. Liability is a most important thing. As yet the average worker is not aware of the fact that he can claim damages and get away with it. There are about 20,000 deaf children in institutes. The annual cost of these institutes is $10,000,000. The ages range from six to sixteen or eighteen years. These children grow up and go out into the world and represent about a third of the totally deaf. There are multitudes of deaf individuals and the time to get them is early and therefore they may be reached in the schools. We ultimately must get

annual examinations for the children in the public schools. We will miss some even then, for occasional ones will develop otitis media a month or several months after the examination. Dr. James A. Babbitt stressed some years ago the fact that much may be done to rehabilitate these patients. Lip-reading may be taught them. Some prevention alone may be possible; 75 per cent are catarrhally deaf. We must stop postnasal drip and must operate the suppurating ears to save hearing. Swelling about the Eustachian orifices keeps up the discharge. The catarrhal ear is at least sometimes catarrh of the canal. In regard to the congenitally deaf we need legislation for sterilization. Usually when the deafened bring into the world a child who is deaf we can convince them of the importance of sterilization. Otosclerotic cases are not capable of being diagnosed early. We need legislation for the deaf because the deaf are more apt to be injured or injure others.

Dr. Edwin H. McIlvain: I am not an otologist and was probably selected to discuss these papers because I came from a noisy place. Dr. Smyth believes that business men and engineers realize what noise is and have a sound knowledge of acoustics, but to them noise means prosperity, silence means depression. They like noise. Probably no action will be taken against noises in industry, except nuisance noises, unless definite legislation is encouraged. Electric welding now eliminates rivetting. Interiors are built so as to eliminate noise. Typewriters are noiseless. Quiet is a good sales argument. Competition may foster this factor. Rubber as foundation for machinery and sawdust compounds have been in the patent office for some time. Mufflers for engines are much the same as the Maxim silencer for rifles. The Maxim silencer and the Burgess muffler are the two outstanding attempts to eliminate or reduce industrial noise. During the past sixteen years I have examined several thousand sheetmetal workers and, eliminating the boilerworkers, I have not over a dozen records of men who complained of ear troubles. They probably did not know they had ear trouble. Noise is a factor in fatigue. This factor must be referred to the psychiatrist and neurologist.

Dr. Walter S. Cornell: In public-health work, in which you are making examinations, it is imperative to have every member of the medical profession working together. Many cases are not treated because of disagreement of the physicians. Differences of opinion are mainly met in considering the question of eye strain. The examiner finding deafness sends a note and the family doctor minimizes the condition and blocks the effort. Expensive consultants may also be a factor in discouraging further treatment. The school examination should be carefully considered. It is a pity that we are allowing the sociologists to take the lead on these questions. Noise is not only a factor in industry but also in the general environment, and the factors of light, heat, chemicals, dust, etc., must be considered seriously. As risks multiply, public-health workers are striving to correct these conditions as fast as they are found. On the question of compensation, it is pitiful to see the physical condition of men of fifty who apply for the position of school janitors. The city or state should take men 50 per cent or 60 per cent efficient and give them a suitable job.

Indications for Removal of Tonsils and Adenoids

Dr. George B. Wood: There is no doubt but that the removal of the faucial and pharyngeal tonsils is frequently followed by most beneficial results, and so much so that it has come to be a most popular procedure, probably outranging, twenty to one, all other operations on the nose, throat and ears. Its popularity is not restricted to the medical profession, as modern parents do not consider their child's education complete until this mutilation has taken place. It is possible that some day we will have on the statutes of our various state legislatures laws making this operation compulsory before a child can be admitted to a public school. In fact, I have had pediatricians intimate that such a thing might not be a bad idea. If the tonsil were a functionless organ, just a vestigial remnant, as is the appendix, and if the operation were free from danger and was not accompanied by pain and discomfort, one might advisedly look to the legislature for aid in this time of financial depression.

However, from their often-expressed opinions, it is evident that experienced laryngologists do not concur in the idea of the prophylactic operation and recognize the fact that the tonsillar structures of the throat may at times be perfectly normal in that they are not a present menace to the health of the individual. In other words, that there are certain phenomena which should be considered as indications for the operation, although it is probable that what constitutes an indication varies considerably in the minds of different operators. The tonsillar tissues of the throat do have in early childhood an important function as a member of a large group of hematopoietic organs; that the operation is not devoid of pain and discomfort and that it is attended by enough fatalities to class it as an almost major operation, must also be admitted.

Indications for the operation in children differ somewhat from those of adults, because in children the removal of the faucial tonsils is often done as a sort of an appendix to the removal of the adenoids, whereas in the adult the pharyngeal tonsil is seldom disturbed. In not a small percentage of children, the early removal of the faucial and pharyngeal tonsils is followed by hypertrophy of the other lymphoid tissues of the throat and this hypertrophy may become a very distinct menace to the patient. It is exceedingly difficult to deal with this diffuse tissue. In recent years it has been customary

to limit the operation to those over five years whenever the indications were not urgent. It was surprising to find that indications which were thought present between two and three years of age had disappeared two or three years later.

If a positive history of recurrent tonsillitis has been obtained, the operation is indicated without regard to the local appearances of the tonsillar structure. This also holds true in regards to peritonsillar inflammations. For clinical purposes the cervical lymph nodes may be classed in two groups, the superficial and deep, but it is the deep glands that especially interest us in the study of throat infections. These are divided, anatomically, into the anterior and posterior groups by the anterior edge of the sternomastoid muscle. Situated where the posterior belly of the digastric crosses the anterior margin of the sternomastoid muscle is the largest of these anterior glands. We may term this the tonsillar lymph node, because it receives the lymphatics from the faucial tonsils and the surrounding mucosa. The glands that lie under the muscle constitute the posterior group and receive drainage from the pharyngeal vault and nasal fossæ. Therefore, in an infection originating from the pharyngeal tonsil, we would expect that the group underlying the muscle would be enlarged, whereas the infection from the faucial tonsil would cause enlargement of the so-called tonsillar lymph node. Chronic adenopathy of the tonsillar lymph node and neighboring glands results from faucial tonsillar infection in probably 90 per cent of the cases. If this adenopathy is marked, if it shows recurrent exacerbations, we have a fairly strong indication for tonsillectomy.

Adenoidectomy is indicated between attacks of otitis media where the pharyngeal tonsil is enlarged or when it has been acutely inflamed at the time of the otitis. Faucial tonsils bear little etiological importance to the infection through the Eustachian tube unless they are greatly hypertrophied or are badly embedded high up in the palate. Undoubtedly a pharyngeal tonsil that is large enough to produce mechanical obstruction has a causative relation to nasal infection, including sinus involvement; also, in children with frequent colds careful examination of the pharyngeal tonsil should be made at the beginning of these colds, as in certain cases the infection originates in this lymphoid tissue of the nasopharynx. Faucial tonsils do not have an etiological bearing on nasal infections and we are not justified in doing faucial tonsillectomy for nasal

infections and its associated complications. When enlargement of the faucial and pharyngeal tonsils is great enough to provide mechanical obstruction, their removal is indicated. The faucial tonsil without other indications would need to be exceedingly large to warrant its removal, whereas the pharyngeal tonsil must be frequently removed just because of its size.

Outside of operations done for the relief of acute infection of the tonsil itself, it is possible that systemic disturbances resulting from the absorption of toxic materials from the tonsil is responsible for more tonsillectomies than any other series of indications. This is especially true in adults. It is exceptionally difficult to make a prognosis in these cases and there is no method of examination that will determine with certainty the responsibility of the tonsil for any systemic infection. The number of diseases that are supposed to be of focal infection origin is so great that they could not be possibly covered individually this evening. Two or three of them seem to occupy considerable attention and perhaps merit discussion. Rheumatism and its allied conditions are today etiologically considered focal infection diseases. In acute rheumatic polyarthritis and secondary chronic polyarthritis, the tonsils, whatever their appearance, should be removed when the acute symptoms have subsided. In cases of rheumatoid conditions and chronic infectious rheumatism, in neuralgia and myalgia, the tonsils should be removed if diseased. In osteoarthritis, Bechterew's disease, gout and arthritis deformans, tonsillectomy seldom, if ever, is of any value.

Bacteriological studies of the tonsillar flora have failed to produce much data that is of value. The pressing out of cheesy or purulent crypt contents may be done in perfectly normal individuals, but it must still be considered evidence of tonsillar responsibility in most cases. The purulent débris is of more significance than the harder cheesy débris, which may be only desquamated epithelium. The mere finding of a small quantity of pus in some of the crypts of the tonsil is not by itself an indication for tonsillectomy. As a rule, large tonsils are more frequently sources of harm than smaller ones, but the smaller tonsil can at times be a very serious menace. Narrowed crypt mouths or small whitish areas on the surface of the tonsil, due probably to encysted collections of pus, are important local findings. Yet tonsils with a perfectly normal appearance may give rise to serious constitutional disturbances, and many healthy and normal individuals have the most foul-looking tonsils. In

rheumatic cases tonsillectomy is not a complete prophylactic guard against more serious complications. Statistics would indicate that tonsillectomy had no effect in the prevention or cure of chorea, nor in preventing any of its complications.

It is not the duty of the laryngologist to judge in individual cases whether the presenting symptoms are a disease of focal origin or not. This belongs to the general practitioner or internist, or perhaps to the specialist of other parts of the body, the eye surgeon, the neurologist or the urologist. We need from the internist a statement merely that the condition from which the patient suffers is focal infection in origin, and then it becomes our duty to estimate the importance of the tonsillar tissues in its etiology. In doing this we must take into consideration the history of the case as presented by the internist, with his findings opinion, the history of local throat inflammatory conditions, rigidity separating the purely tonsillar inflammation from other infections conditions of the upper respiratory tract; the condition of the lymph nodes of the neck, as well as the appearance of the tonsils themselves, and after we have done all this we still have to operate on the possibilities, perhaps probabilities, rather than any absolute certainty of relief. Tonsils are not the only source of infection, and it is essential that careful studies of the teeth, sinuses and gastrointestinal tract be made and cleared up if possible before the tonsil operation is done.

Discussion. Dr. Ralph Butler: We have to work on probabilities. Given a case with focal infection, we are unable to tell if abscessed teeth or infected tonsils are the cause. Prognosis of the postoperative effect is not accurate nor wise.

OCTOBER 21

Cerebrospinal Rhinorrhea (Report of Two Cases)

Dr. J. C. Donnelly (by invitation): Two cases of cerebrospinal rhinorrhea were reported: one, the spontaneous, survived; the other, traumatic, died. The first case, that of an asthmatic male, aged fifty-seven years, presented advanced hyperplasia of both middle turbinates; and the middle meati and the left sphenoethmoidal area were filled with polyps. The frontal and right maxillary sinuses were hazy on transillumination; the right antrum was opaque.

Believing the sinus pathology an important factor in the bronchial asthma, bilateral ethmoidectomy was undertaken. When the right middle turbinate was removed extensive polypoid degeneration was found in the posterior ethmoidal cells. Some superficial tissue was removed with a Blakesley alligator forceps. The second bite into the polypoid mass was followed by a thin, watery discharge, which became more profuse and was noted to pulsate. Suspecting the leakage to be cerebrospinal fluid, operation was stopped and the patient sent to bed. While being placed on the ward carriage the patient fainted. On the basis that this was due to a sudden decompression and that the loss of fluid was too great, the ethmoidal area was packed with iodoform gauze. Adrenalin and atropine were administered, and in a few minutes the patient regained consciousness.

Upon his return to the ward the patient was cold and clammy, with a temperature of 97.2° F. and a pulse-rate of 60. The nasal packing was then removed and a policy of dehydration instituted to reduce intracranial pressure and facilitate early closure of the operative wound. To this end fluids were restricted and large doses of magnesium sulphate were given. Twenty-four hours after operation the rhinorrhea ceased. Two days after operation the temperature and pulse-rate remained normal and the patient's strength was such that he repeatedly requested to go home. Seventy-two hours after operation the temperature soared to 103° and the patient complained of severe occipital headaches, chills and nausea. Restlessness and delirium followed. The pupils contracted, but reacted to light. A spinal puncture revealed turbid fluid but no increase in pressure; the cell count was 16,000 polymorphonuclears. Kernig's sign and cervical rigidity appeared and the cell count rose to 35,000. Five days after operation the patient died.

At autopsy the brain surface was covered by a thick, yellowish, purulent exudate which was especially abundant toward the base. The convolutions were distinctly flattened. Hemolytic streptococci were cultured from the cerebrospinal fluid both before and after death. An opening with ragged edges, measuring 1 by 1.5 cm., was found on the right ethmoidal roof, about 1 cm. from the midline or 1 cm. from the lamina cribrosa. This opening led into the ethmoidal cells which were filled with a soft, foul-smelling, gelatinous mucosa. There appeared to be senile atrophy of the bone, but no necrosis. The left ethmoidal roof was likewise atrophic.

The second patient, a woman, aged sixty years, weighing 225 pounds, had noticed a watery discharge from the right side of the nose during an attack of influenza. Recovery from influenza was uneventful, but thereafter she awoke every morning with severe bitemporal headache. At 9 or 10 o'clock the headache disappeared and rhinorrhea began. The flow continued intermittently throughout the day and night, with the exception of the early morning hours, at which time her headache was present. Approximately 12 ounces of fluid were lost in twenty-four hours. Bilateral pressure on the internal jugular veins occasionally produced a gush of fluid from the nostril. Other nasal symptoms were absent and there was no history of injury or operation.

A clear, watery fluid was observed to ooze from the right sphenoidal ostium. A roentgenogram revealed a peculiar appearance of the middle fossæ, the identity of which was uncertain. The dorsum sellæ is quite thin, as if eroded from behind. This may also have caused an erosion into the sphenoidal sinus and would thus account for the cerebrospinal rhinorrhea. The only therapy of proven value has been a reduction in the amount of fluid intake. It is now twenty-one months since the onset of the rhinorrhea, which continues, but the patient is attending to her household duties.

Discussion: Dr. George M. Coates: These cases are rare and infrequently reported. It is disturbing to the ethmoid operator to find that the roof of the ethmoidal structure may be broken by a small tug. We usually suspect a break in the cribriform plate, but in this traumatic case there was an unusual break. When meningitis occurs after ethmoidal operations I often suspect the infection may travel by way of the perineural sheaths. One of my cases, with pituitary enlargement, a typical case of acromegaly, had had cerebrospinal rhinorrhea for seven years and is alive and well. Several drops of fluid are lost per minute and the pathologist found this fluid to be typical of cerebrospinal fluid.

Dr. John E. Loftus: Such accidents must occur to all of us. They are rarely reported. I think it is a mistake to pack after ethmoidal operations, should rhinorrhea appear. A case operated in Germany had a meningocele removed in mistake for a nasal polyp. When the mistake was recognized immediate operation was done to suture the dura and the patient got well. Spontaneous cerebrospinal rhinorrhea gives the same type of history. Tumors and internal hydrocephalus do not always develop cerebrospinal rhinorrhea. The tumor cases in which Dr. Cushing has performed transnasal sphenoidal operations all heal without developing rhinorrhea.

Dr. Warren B. Davis. Any of us who have worked for a long time in the dissecting room recall sphenoids in which definite dehiscences of bone

occurred. Usually these are posterior and lateral or in the superior portion of the sinus. These areas may become weak because of pressure. In a series of sinuses we studied several years ago we found thinning of the bone in a number of sphenoids.

Dr. George B. Wood: A traumatic cerebrospinal rhinorrhea was admitted to the Graduate Hospital several years ago. A colored woman had been struck several times by a hammer in the hands of her husband. The first blow destroyed the lower wall of the frontal sinus, crushed the nasal bone and the anterior ethmoidal cells; the second blow broke and depressed the anterior wall of the antrum; the third blow cut off the external ear and depressed the mastoid area. A profuse cerebrospinal leak developed from the cribriform plate. Dr. Grant cut across the brow, turned back osteoplastic flaps, elevated the frontal lobe and explored the cribriform plate. The site of the leak could not be distinguished, so packing was placed over the cribriform plate. The woman got well and the cerebrospinal rhinorrhea disappeared. If we are daring enough we may save some of these cases.

Dr. Samuel Skillern: The posterior portion of the ethmoid is the dangerous area. Several years ago I got into the dura. I immediately went through the anterior and posterior plates of the frontal sinus, found the hole, packed and waited. The patient died within twenty-four hours. Another case, in which I attempted to sound the sphenoidal sinus after a complete extirpation of the ethmoid on the right side, did not seem to have a posterior sphenoidal wall. I obtained a longer probe, but this did not touch a bony wall. Calling my brother and Dr. Ridpath, we placed a longer probe through the ostium and an *x*-ray plate showed the probe touching the top of the cranium. I expected the usual fatality in forty-eight hours, but the patient made a beautiful recovery and was discharged from the hospital. A year later the patient developed a splitting headache and delirium and died. At autopsy, when the skull cap was removed, 4 to 8 ounces of blood gushed forth. We found a hemangioma of the left frontoparietal lobe which had burst. The blood Wassermann was reported as 4+. A probe could not be pushed through the dehiscence and no vestige could be found of the opening through which the probe had been previously introduced.

Serious Complications Following Tonsillectomy (Case Reports)

Dr. George B. Wood: A male, aged four years, was admitted to the hospital with a chief complaint of swelling of the left knee beginning ten days after smallpox vaccination. A slight, irregular fever continued until the knee was incised, but five days after operation the temperature became normal. Three months after admission tonsillectomy was considered, with the view of removing all sources of infection before discharging the patient. A rise of

temperature to 102° for twenty-four hours at this time was due to a slight infection of the tonsils. The tonsils were large, but otherwise negative. The paranasal sinuses were negative. A fluoroscopic examination of the lungs showed a slight increase in density in each hilum, but not sufficient to suggest tuberculosis, and the Von Pirquet test was negative. Fluid from the knee was sterile and failed to kill an inoculated guinea-pig. The white blood cell count varied from 7000 to 15,000, with a slight excess of lymphocytes. The urine was normal.

In accordance with usual technique, sodium bicarbonate was administered for twenty-four hours before operation, with a high carbohydrate diet, cleaning the teeth thoroughly and inducing moderate evacuation of the bowels. The tonsils and adenoid tissue were removed under ether anesthesia, and the child left the table in good condition, with dry throat and a pulse-rate of 112. Three and a half hours after operation the temperature was 99.2° by rectum, but six hours later had risen to 102°. Fifteen hours after operation the temperature reached 104° and three hours later 105°, reaching later 106.2°. The pulse-rate increased as the temperature rose, its highest rate being 150. There was no active bleeding after the operation. The child was rational until two hours before death, which occurred twenty-four hours after operation. Just before the exodus the child became cyanotic. There were no definite signs of pneumonia. The throat showed a certain amount of edema about the uvula and the patient complained of a good deal of pain in it. There were no neurologic symptoms until an hour and a half or two hours before death, when some rigidity of the neck and spine appeared. No autopsy could be obtained.

The cause of death in these cases with severe postoperative hyperpyrexia is extremely difficult to explain. There is a close resemblance between these cases and those in which there is some lesion of the diencephalon, where the theoretical heat center is located. There are clinical examples in which attempts at removal of suprasellar tumors which encroached on the base of the third ventricle were followed by a gradual increase in temperature, with death in two or three days; also in certain cases of fracture at the base of the skull there is marked hyperpyrexia. Further, hemorrhage into the third ventricle, or an embolism which involves this area of the brain, would produce a similar clinical picture. It is scarcely conceivable that any surgical trauma in the region of the

faucial and pharyngeal tonsils could in any possible way directly injure the brain stem. As far as the bloodvessels are concerned, there is no direct connection between the pharyngeal vault and the suprasellar region, so that the embolic theory is scarcely tenable. The only possible anatomical connection would be, perhaps, a persistent Rathke's pouch, the embryologic anlage from which the anterior portion of the pituitary body is formed, and originating from the nasopharynx. However, these cases of postoperative hyperpyrexia are not always results of tonsil operations, but they have been noted in children following a variety of surgical operations.

Dr. Karl M. Houser: The patient, a female student, aged eighteen years, was admitted for tonsillectomy. At the age of five years the tonsils and adenoid tissue had been removed, but the tonsil removal was incomplete. Because of recurrent attacks of sore throat during the winter months she was advised to have the residual tissue removed. At 2 P.M. 3 grains of sodium amytal were administered and one hour later the operation was performed under procaine anesthesia. No cocaine was used previous to the injection, and for injection the carpule injection syringe was used, so that there was no possibility of cocaine having been injected by mistake. In all, two carpules of 1 per cent procaine and epinephrin were used. The operation was well tolerated by the patient, she was at no time unduly nervous or apprehensive, nor did she show any tendency to syncope. The tonsil masses were removed with little difficulty and very slight bleeding. On her return to bed the temperature, pulse and respiration were normal. The last recorded temperature, pulse and respiration, six hours after operation, indicated both pulse and respiratory rates were normal, with a slight elevation of temperature to 99.3°.

At 10 o'clock a resident was summoned on account of slight bleeding. A blood clot was found in the right fossa and removed. A sponge soaked in 1 to 1000 adrenalin chloride solution promptly controlled the bleeding, which was very slight. At 2 A.M., eleven hours after operation, bleeding was again reported. A clot in the right tonsil fossa was found and removed and a sponge soaked in 10 per cent cocaine hydrochloride and adrenalin chloride solution applied for about one minute. This was followed by an application of Monsell's solution. A small bleeding-point was then discovered and clamped with a hemostat. Almost immediately a generalized convulsion occurred and respiration ceased. No radial pulse could

be detected. Adrenalin chloride subcutaneously and caffein sodium benzoate were immediately given, along with artificial respiration. Later adrenalin was injected directly into the heart and oxygen administered, along with cardiac massage. Death occurred in spite of all efforts. This case supports the work of Dr. Emil Mayer in his various reports upon toxic manifestations following the use of local anesthetics, and should be recorded as a warning to all against the dangers of using cocaine preparations in any concentration in throat surgery.

Dr. Edward H. Campbell: The case reported was that of a female, aged thirty-five years, who developed cavernous sinus thrombophlebitis following tonsillectomy under local anesthesia. The anesthesia consisted of the infiltration of the peritonsillar area with 1 per cent procaine solution, which contained a few drops of epinephrine. The tonsils were removed by the dissection and snare method. The day following the operation the temperature was 100° F., but there were no other unusual symptoms at this time. On the second day after operation a severe chill was followed by a rise in temperature, and a swelling in the right peritonsillar region suggesting an early abscess. The abscess was incised the following day, with the evacuation of thick greenish pus containing Gram-positive staphylococci and Gram-positive diplococci. The temperature at this time varied from 99.4° to 100.3° F., and the leukocyte count was 17,700, with 82 per cent neutrophils.

The throat symptoms improved after the incision, but the following day pain and tenderness developed in the left temporal and parotid region. Twenty-four hours later edema, first of the right eye and then of the left, was noted. Tenderness and swelling of the left side of the face increased and extended downward over the parotid region, but no fluctuation developed. There was swelling in the left peritonsillar region, but not sufficient to indicate an abscess. There were daily chills followed by a moderate rise in temperature, and a blood culture at this time contained nonhemolytic streptococci. Swelling of the eyelids rapidly increased and a distinct proptosis was evident. Paralysis of the right external rectus was noted and other ocular movements were faulty, but no swelling of the optic disks or hemorrhages occurred. Ten days after operation the swelling of the left side of the face had developed to such an extent that, although no distinct fluctuation was evident, a deep incision was made in the preauricular area, releasing a small

amount of pus and very dark-colored blood. The following day the incision was enlarged and deepened, without finding an abscess pocket. General stimulation, frequent infusions of glucose and saline and blood transfusions were employed, but the ocular signs of cavernous sinus thrombosis steadily progressed. Both eyelids became more edematous, the exophthalmos steadily increased, considerable ecchymosis developed and the eyeball itself became immovable. The temperature reached 107° F. and the patient died on the thirteenth day after operation.

A partial autopsy was permitted and the incision on the left side of the face was explored. A considerable amount of seropus was expressed from the tissues, but no localized abscess pocket could be found. Sections of the lungs showed multiple hematogenous abscesses; the heart and liver showed cloudy swelling and the kidneys gave evidence of acute tubular necrosis. Unfortunately, the brain could not be obtained, but the typical signs and symptoms of cavernous sinus thrombosis (thrombophlebitis) indicated plainly the diagnosis.

Discussion: Dr. B. H. Shuster: One of my cases, a baby, aged two and a half years, barely escaped death after tonsillectomy. The child went home the day after operation and later in the day I was called and told that the baby was falling asleep and could not be aroused. No food had been taken for a day and a half. A catheterized specimen of urine showed sugar and acetone. The patient was filled with fluids, and glucose and alkalies were administered and the patient survived. Dr. Campbell's case probably came about by thrombosis by way of the pterygoid plexus, but we may also get thrombosis of the internal jugular vein.

Osteoma of the Paranasal Sinuses (Case Report)

Dr. J. Austin Smith: A male, aged sixteen years, came to the Jefferson Clinic complaining of marked swelling, redness and tenderness over the left frontal sinus. At this time the left eye was closed by the swelling and edema of the surrounding structures. A small, hard lump on the left side of the nose at the inner third of the floor of the left frontal sinus had first been noted by the patient two years before coming to the clinic. It was not tender at that time and gave him no discomfort, but gradually increased in size and later there was swelling of the forehead and the lump encroached upon his eye. A month before coming under observation the patient was in a fight and was struck heavily at the site of the growth.

Upon examination, the patient presented the picture of acute frontal sinusitis, with perforation and involvement of the soft parts. The eyelids were closed by the surrounding swelling and edema, and there was marked tenderness with fluctuation over the swelling above the eye. The nasal chambers were clear and ventilation and drainage appeared good. To secure drainage an incision was made beneath the left brow and considerable pus was obtained. The cellulitis subsided until the tumor mass could be readily palpated. No attempt has been made to remove the growth. The roentgenographic studies reveal a lesion involving the left frontal sinus in which there is a mass with density almost that of bone. It apparently projects into the orbit. The appearance is that of an osteoma. The direct posteroanterior films show that the left frontal sinus is completely filled with the tumor mass and it is extending into the right frontal sinus, as well as into the left orbit. (Roentgenograms and patient presented.)

Discussion: Dr. Robert J. Hunter: I have a case under observation which came to our attention four years ago. The tumor appears more dense in the roentgenograms than does the one reported here tonight, and measures about 2 to 3 mm. in diameter. The patient, a young lady, now returns because of a mild cold. The osteoma appears to be the same size as four years ago.

Dr. Warren B. Davis: About six or eight years ago Dr. Shallow asked me to assist him in an operation upon an osteoma of the orbit, involving the ethmoidal cells and the antrum. There was no sinus infection. Through a radical frontal incision a mass of dense bone was discovered, rather spherical, about $\frac{3}{4}$ to 1 inch in diameter. The eyeball was displaced to facilitate removal of the tumor and the lower portion of the orbit plate and the ethmoidal plate were removed. The eyeball returned to its position and there has been no recurrence to date.

Dr. Philip Stout: I saw a case of osteoma of the frontal sinus about a year ago. Six months later a roentgenogram showed it to be about the same size as formerly. These tumors sometimes respond to radiation.

NOVEMBER 17

A Short Résumé of the Results of the Ballance-Duel Research in Nerve Anastomosis and the Employment of Nerve Grafts in the Surgical Treatment of Facial Palsy (Moving Pictures and Lantern Slides).

Dr. Arthur B. Duel (by invitation): Dr. Ballance had performed his first anastomosis of the hypoglossal nerve to the facial nerve in

1895. His important work on the anatomy and surgery of the temporal bone was widely recognized twenty-five years ago. A year after the World War Sir Charles Ballance came to the United States, and during his visit suggested to Dr. Duel that they retire to a quiet place, where animal experiments could be carried out, to devise an operation for the cure of facial palsy without associated movements to mar the result. A laboratory was built in the country which eventually accommodated 40 large apes, 70 to 80 cats and 25 large Louisiana frogs. Sir Charles Ballance superintended the building of the laboratory, and when that was completed confined himself entirely to the work in hand, working with marvelous industry from day to day, without exercise or sufficient rest.

Various types of anastomoses were considered. Patients with an anastomosis of the spinal accessory and facial nerves were unable to use the facial muscles without accessory movements, but a large number of similar anastomoses were done to verify previous experiences. Anastomoses of the descendens noni, glossopharyngeal and chorda tympani nerves with the facial nerve were attempted. In every instance where such anastomoses were done the facial canal was opened, plugged with gold and carefully inspected, because in Sir Charles Ballance's experience the stump of the facial nerve was in such close proximity to the anastomosis that there was a question whether or not tissue had leaped across the scar by residual strands and thus be suspected of influencing results. In the smaller animals the descendens noni and the hypoglossal nerves were anastomosed with the facial. Then the auriculotemporal nerve was joined to the facial in larger animals, utilizing a nerve which could show no associated movements but failed to produce the finer movements obtained when the hypoglossal or spinal accessory nerves were used.

For a long time Sir Charles Ballance had advocated taking the facial nerve out of its canal and running it across the tympanus to get length. The technique required to accomplish this without trauma was carefully developed. The great occipital nerve for anastomosis with the facial precluded associated movements. The great auricular nerve was likewise tried for this reason.

In one infant, with the facial nerve largely destroyed by a mastoid operation, one end of the nerve was situated in the parotid gland and the other end presented at the geniculate ganglion. Recovery followed the grafting of the facial proximal portion to the facial

distal portion. In an adult female 16 mm. of the facial nerve had been removed in a radical operation, resulting in complete palsy. A portion of the nerve of Bell was placed in the dehiscence four and a half months after operation and this patient has some ability to close the eye.

A baboon, in which the intercostal nerve was used for anastomosis with the facial nerve, showed complete recovery. In another patient the nerve of Bell could not be located at operation, while a large cutaneous nerve was uncovered and an attempt was made to connect it with the facial nerve. Its length was not sufficient but the workers felt that it would work out as well as a motor nerve. Making certain that the distal portion of the nerve was alive, a portion of the third intercostal was placed in the canal but its length appeared insufficient. A piece of the fourth intercostal nerve was then taken and placed alongside the fragment of the third intercostal nerve and the whole covered with dental gold. Six days later the patient obtained the sensation of movement.

No one can predict the degree of recovery in these cases. The animals used in this series of experiments with grafts have shown recoveries which are practically perfect. Twenty-four animals have shown recovery from dehiscences of 1 to 16 mm. Motor and sensory nerves were both used with an equal amount of recovery. A child, aged eight months, shows recovery which is progressing and at this time the normal impression of pleasure or crying is almost perfect. If man were like an animal almost perfect recovery could be obtained; in man it is difficult to ever restore a divided or injured facial nerve and obtain a result which synchronously shows the higher emotions. It is difficult to get the finer gradations of expression through a repaired nerve. In order to find the anastomotic tissue we get trauma and scar tissue and, therefore, produce interference with synchronous effects. Fear and laughter may be well done but the finer gradations of emotion will be different from those obtained before injury occurred.

The operation as perfected will restore the facial muscles and avoid the associated movements which in other operations can be eliminated only at best by training. In addition, this operation avoids a disfiguring scar in the neck and the loss of function of other muscles. Any anastomosis operation is difficult and this is no exception. No one will be able to attempt it without giving time enough to master the technique.

Discussion. Dr. Charles H. Frazier: It is at once stimulating and refreshing to hear of this distinct achievement. The operation described here has been performed on man and animal and a treatment has been elaborated which is a distinct step forward in the therapy of facial paralysis. The repair of damaged nerves is not unfamiliar to me. In one center during the World War we had 1200 such cases and I felt rather fed up on operations involving the peripheral and cranial nerves. I would like to confine my remarks wholly to facial paralysis, and to review some of my observations. When should the operation be performed after a facial paralysis? Our rule is to wait six months but one cannot be too arbitrary. One day after six months one of my patients had a quiver at the angle of the mouth. Another patient, a woman with a tumor of the parotid, began to recover one year after operation. A peculiar sensation foretells recovery; a sense of power appears before the patient is able to demonstrate power. The time of operation depends upon the lesions, that is, whether the injury is due to a mastoid, tumor or other cause. Another problem frequently presented is, "How long can an operation be deferred before operating?" In one case twenty-two years had elapsed before operation, which was followed by reasonable success. I always tell my patients what they may expect if we use the hypoglossal or intercostal nerves—that they will have facial expression, will be able to close the eye, but will not be able to use the occipitofrontalis, which usually is the last to appear and sometimes never returns. I also tell patients that if they take the time and the trouble they can again smile if they practise the act with a mirror. We frequently first notice a difference when the patient smiles. The return of emotional expression must not be anticipated. Even in return by spontaneous recovery, movement on the affected side is not synchronous with that on the sound side. There is also a difference in voluntary and involuntary movements.

Associated movements are recognized as an objection to anastomotic operations. I have never found in my patients that hypoglossal movements were very disturbing. Patients frequently ask you which nerve you intend to use. If you say hypoglossal, they may say, "Then half of my tongue will be paralyzed." I say, "Yes, but only one-half of the tongue will be affected and speech and deglutition will not be affected." With the descendens noni there are no associated movements to embarrass the patient. In discussing the use of nerve transplant we should remember that bridging of the defect is not new. Dr. Huber, working in governmental laboratories during 1917, 1918 and 1919, tried many ties of anastomoses and grafts. In reviewing Huber's original article I found that the length of his grafts were 3 cm. We hoped after this publication that a panacea for gunshot wounds had been obtained by posture, rerouting the nerve or by stretching, but found that over 6 to 10 cm. the situation seemed hopeless and a transplant was needed. Grafts in shotgun and shrapnel wounds were found to be almost hopeless and usually a total failure. In 1919 I used grafts in two cases and secured regeneration, but these were exceptions rather than the rule.

A graft, 3 cm. in length, restored function in the experimental laboratory;

in human beings a graft of 10 cm. or more did not restore function. It is, therefore, not surprising that in the facial canal, with a defect of 2.5 to 3 cm., results were gratifying. When damage is done during a mastoid operation we should probably repair the injury by direct suture rather than by a graft.

Dr. William C. Spiller: I presume, of course, that in using grafts experiments have been done in transferring nerves of one animal to another. For example, transferring a nerve from an ape to a gibbon. It is not advisable to use a nerve from an animal for a graft in man. Sir Charles Ballance is the greatest authority in the world on nerve anastomoses. His first case gained prominence, in 1895, and he has carried on his work through all the years since then. He has, however, surpassed himself recently by grafting a sympathetic nerve to the peripheral facial stump. He reports that at the ninth month in one of his animals the face was symmetrical, and at the eleventh month, by exposing the brain and applying an electrode to the appropriate area, muscular contractions were obtained in the facial distribution. We can understand how it is possible to get motor function with the glossopharyngeal and spinal accessory nerves, but in dealing with the sympathetic nerves we have no evidence of their representation in the brain cortex. Voluntary movements have not been demonstrated to be absolutely normal in these operated animals. Sir Charles Ballance once asked me what the condition of the cerebral cortical cells in the region of Rolando would be after the facial nerve was cut so as to destroy 6 to 12 mm. of its substance. This would depend on the age of the patient. When a nerve is destroyed in a young animal the cells in the cortex representing those nerves may disappear; in older animals the cortical cells may survive. Cells of the first neurone are probably not damaged when the cells of the second neurone are destroyed.

Dr. B. Alexander Randall: A case I have mentioned several times had facial neuritis following a mastoid operation. There was stiffening of the face and increasing loss of power but no paralysis. The condition disappeared after many months but some residual symptoms were noted three years after operation. Mere digital manipulation and massage (after the neurological department thought there was no possibility of recovery) brought about the improvement. Later function was again lost with the appearance of middle-ear infection and spasticity became permanent. Successful results may be good temporarily but some of them may not be permanent.

Dr. James A. Babbitt: I asked Dr. Duel what had been developed in these research studies of a practical nature. The substitution of this new method seems to provide results without the associated movements. The technique is practical but seems to be essentially that of the skilful neurosurgeon.

Dr. Robert J. Hunter: Facial palsy sometimes precedes the mastoid operation and after operation many of these recover. Could Dr. Duel's and Sir Charles Ballance's method be applied to such cases?

Dr. Abraham Ornsteen: I note that in two of Dr. Duel's cases there were different results. The adult female had had paralysis for a year

before operation was attempted. An infant was operated upon almost immediately after injury. The time element, therefore, seems important. In the adult there is little recovery in the lower face. The animals operated upon were done shortly after dehiscence had occurred and conditions were, therefore, favorable. Has Dr. Duel seen favorable results in animals when the injury has been of long duration? We cannot expect good results when there is a long peripheral stump. This operation, in order to secure good results in traumatic mastoid cases, should be done as early as possible.

Dr. Duel: The point raised by Dr. Ornsteen may be answered by a point brought out by Dr. Frazier. He spoke of the premonition a patient has of a sense of power in the face. In our animals we traumatized the nerve and there was no opportunity for degeneration of muscle for the reparative process began at once by the introduction of the graft. In the infant I report repair was made at once and, although there was reaction of degeneration, yet recovery was about as rapid as in a Bell palsy. The longer the delay before repair, the greater the degeneration of the fiber and muscle, and the less favorable the prognosis.

When the response to galvanic electricity occurs in the muscle repair of the dehiscence will be followed by recovery, but when the response is poor to galvanic currents the prognosis is poor. Two adults mentioned the sensation Dr. Frazier has described. It is the certainty of something about to happen in the involved area. Trophic influences appear before the muscle actually functions. The reason why apparently perfect recovery occurs in animals while less perfect recovery occurs in human beings, has been given by Hunt, who describes different levels from which stimulation comes—paleokinetic influences, which spring from lower levels, and have to do with essential movements such as those of the face in eating, and neokinetic influences, springing from higher levels, and dealing with the expression of emotions (response to music, speech, etc.). Neokinetic influences are probably only present in human beings.

As to the question of the time of operation on a nerve after injury, Sir Charles Ballance advises, "Do it now; do not wait." Poor results may be gotten by instituting medical treatment before trying surgery. In trauma there is pressure on a nerve. Therefore, uncover it so that permanent loss of function may be prevented. With proper technique, we will not do as much permanent injury to a nerve as by waiting.

DECEMBER 15

Motion Pictures of the Larynx

Dr. C. A. Heatley (by invitation), Rochester, N. Y.: In presenting a short film of motion pictures of the larynx, the important possibilities of laryngeal photography in the teaching of laryngology as well as in recording rare cases, were stressed. There is great

difficulty in photographing small objects. In 1883 Lenox Brown took the first photograph of the larynx, but he and his coworkers saw no future for photography of the larynx. Thomas French, in 1884, produced photographs of really surprising merit by attaching a camera to a mirror and reflecting sunlight toward the patient's larynx by means of a system of truncated cones. Later he developed a powerful arc light which accomplished the same end. This work was forgotten until 1913, when Garel produced the stereoscopic camera. Dr. Clerf has used this camera sufficiently to be most familiar with its intricacies. Most of the methods developed are too technical to appeal to most laryngologists. The teaching possibilities of the motion picture have come into prominence in recent years. Russell, in 1930, obtained small pictures of the larynx, but he was more interested in phonetics than in pathology.

The apparatus used to obtain motion pictures of the larynx was shown in lantern slides. The chief problem is that of constructing an apparatus which may be used by any laryngologist. In this instance an ordinary direct laryngoscope was used with the patient under local anesthesia. The instrument is purely experimental and was constructed without support from any manufacturer. A model B Cine-Kodak is placed on the opposite side of the lens. A side lens gives control of the field on the end of the laryngoscope. The greatest difficulty lies in securing adequate illumination of the larynx. This instrument focuses the rays to the end of a quartz rod which carries the light directly to the end of the laryngoscope and the light is virtually sprayed over the larynx. About 400 to 500 foot candle-power is obtained. The lens and the film back of it are centered on the axis of the laryngoscope.

The film itself demonstrated the normal larynx, paralysis of the left vocal cord secondary to an aneurysm and early carcinoma of the vocal cord. These photographs prove that laryngeal motion picture photography is feasible and practical. The intensity of the illumination may be readily increased and more sensitive films used. Color photography may be possible with greater illumination but this will require 25 to 30 times the intensity of light used in the film shown tonight. Color photography is not practical at the present time.

Discussion: Dr. James A. Babbitt: Did the investigators discover anything by this type of photography that the ordinary examination would fail to discover?

Dr. Lewis H. Clerf: Since I have worked with the Garel instrument, I can especially appreciate what Dr. Heatley has accomplished in these films. Their lighting system is a distinct contribution. The larynx is inaccessible, but the main handicap in photography lies in the fact that we are trying to take a picture of a moist surface. It is difficult to show the anterior and posterior portions of the larynx at the same time with the direct laryngoscope. Dr. Healtley has presented a real bit of pioneer work.

Dr. Edward B. Gleason: These are beautiful pictures and good enough for diagnostic purposes.

Dr. Robert F. Ridpath: I have several times tried to photograph the larynx by means of a motion picture camera but never was successful. Supersensitive film will undoubtedly aid further work. I would like to ask Dr. Heatley what type of lens was used. Color film which will be put on the market by the British within the next six months will make color photography of this type possible.

Dr. Nathan P. Stauffer: I would like to ask Dr. Heatley how he fixes his camera on the laryngoscope. With polychromatic film we can get as good pictures as with black and white film.

Dr. Heatley: We did not see anything in our photographs which we had not previously observed in direct laryngoscopy. Such photography is not yet good enough for finding new facts. The camera is fixed on an aluminum plate and the laryngoscope is similarly fixed to the plate so that the whole apparatus is passed as one unit. Both are fixed to the central plate to form a unit. Dr. Clerf has brought out the chief objection to the direct laryngoscope, that of its interference with function of the epiglottis. The other method of approach would be better.

Dr. Henry Dintenfass: The difficulties as I see them are the necessity of a wide open buccal cavity, the necessity of the proper amount of light, the prevention of the inhibition of motion of the vocal cords by the simple laryngoscope. This instrument I am exhibiting, devised by Russell and Tuttle, has a tendency to eliminate Dr. Clerf's objection. With this instrument one can see the movements of one's own vocal cords.

Motion Pictures of Radical Frontal Sinus Operations

Dr. Samuel Skillern: The modified Lothrop, or Skillern-Lothrop operation, was demonstrated. The eyebrows are cut or trimmed by means of a scissors instead of shaving them, to prevent later excessive growth of the hair. The incision is made in the hair line and the periosteum carefully elevated with the overlying soft tissues. It has been found easier to elevate the periosteum in this fashion rather than separately. The sinus is opened above the superciliary ridge and sufficient bone removed to obtain a good exposure and destroy any necrotic area. In bilateral procedures a similar incision is made on the opposite side and the medial ends of the two

incisions joined across the root of the nose. The anterior tip of the middle turbinate is removed and the frontal duct rasped out. This passage is later cleared of diseased tissue and spicules of bone by pulling a gauge strip back and forth. The modified Lothrop procedure is preferable because it requires less operating time and less anesthesia and produces less shock. Constant pressure on the orbit during the Killian operation results in edema of the eyelids and danger to the optic nerve, while there is ever danger of injury to the lachrymal duct. Septal cells as well as small supraorbital cells may be overlooked.

All diseased tissue must be removed to get results. An important step in the operation consists of the beveling of the upper edge of bone. This beveling is a factor in preventing later deformity. The accurate approximation or even oversuturing of the periosteum is likewise important in preventing later deformity. It is better to actually overlap the periosteum in closure to ensure integrity of this area. Complete regeneration of bone may be expected if these precautions are observed. In bilateral cases as well as in unilateral cases the interfrontal septum is removed. This removal in unilateral cases permits aëration of the diseased sinus by way of the normal sinus, and there is no danger of infection of the normal sinus if it has normal ventilation. This frequently leads to discovery of infection in the supposedly normal sinus and it may then be handled at once without subjecting the patient to further operation. The important advantages of this operation are the excellent exposure obtained and the minimum of scarring that is noted later.

Discussion: Dr. Nathan B. Stauffer: I congratulate Dr. Skillern for his beautiful pictures. His reason for the little scarring is the retention of the periosteum. I get scarring anyway. I think he could have done a modified Killian operation in this case and there would then have been less scarring. I like to clean out the ethmoid, sphenoid and antrum at the same time.

Dr. Edward B. Gleason: I congratulate Dr. Skillern on his results in this case. It was difficult to demonstrate any scar. In the motion picture we have a method of teaching operative technique. The best method of becoming proficient is to serve a long apprenticeship. At the operating table the observer is apt to have his attention drawn to the anesthetist, the assistant or other visitors and the operator may be too busy to demonstrate. Motion pictures show more than does the actual operation for in arranging the film attention may be focused upon the important features of the procedure. In the dissecting room we can demonstrate technique a little better than in a motion picture.

Dr. George M. Coates: This type of operation is a much easier operation to perform than the old Killian in many ways and is easier to care for afterward. In this operation the bony walls of the duct are preserved and the pulling of the gauze back and forth is an important step to clear out the spicules of bone and débris. The modified Killian operation, where the external plate is left intact, has the disadvantage in a big sinus of not permitting exploration of the whole sinus and care is necessary afterward to keep the passage open and often there is danger of diplopia.

Dr. Karl M. Houser: What type of incision is used if only one side is opened?

Dr. Skillern: In opening one side, the first incision I showed is the one used. We can expose the whole sinus by undermining. The reason Dr. Stauffer gets sinking-in may be that the periosteum is not permitted to overlap enough. The disadvantage of the Killian operation is that it is terribly disfiguring. The Lynch operation is excellent for a small frontal sinus. We cannot look around edges and angles or through bone. The best plan is to expose everything.

Motion Picture Demonstration of Radical Maxillary Sinus Operation Under Local Anesthesia (Caldwell-Luc)

Dr. Henry Dintenfass (by invitation): The technique used has been essentially the same employed during the past five years. Carpules containing 2 per cent novocaine with epinephrine are employed. Care is employed to infiltrate so that overlapping fibers are reached. Anesthesia of the nasal mucous membrane is induced by the local application of cocaine and by the injection of the carpule mixture into the canalis incisivus and canalis sphenopalatinus. An incision is made at the junction of the labial and gingival mucous membrane and the periosteum with the overlying tissue retracted. An opening is made in the area of thin bone of the canine fossa and enlarged to permit a view of the interior of the antrum. The diseased mucous membrane and polyps were removed from the antrum and an opening chiseled in the nasoantral wall. The cavity was packed with iodoform gauze which passed into the nose through the nasoantral opening and the wound closed by a single suture. It was evident that the procedure was without pain.

Discussion: Dr. Robert F. Ridpath: I have done a number of Caldwell-Luc operations under local anesthesia but always get into something I did not know was there. I ran into so many conditions not diagnosed before operation that I now prefer general anesthesia for these cases. I notice that there was practically no bleeding in this case. I usually get lots of bleeding. Not long ago I operated upon a case which proved to

be sarcoma and the very next case was also a sarcoma, although we did not suspect it of being a sarcoma. I now use avertin in most of my cases.

Dr. George M. Coates: I would like to ask Dr. Dintenfass what percentage of his cases have numbness of the teeth after operation. The vertical incision has been recommended to prevent this.

Dr. Dintenfass: The technique of local injection may be limited to two points of entry, the second division of the maxillary and the infraorbital on the other side. These take care of all overlapping fibers and only two injections are necessary. With small incisions the numbness is not apt to occur.

Motion Picture Demonstration of Obstructive Dyspnea: Bronchoscopic Observations

Dr. Gabriel Tucker: Obstructive dyspnea may be defined as dyspnea due to obstruction in the airway. It may be classified: (1) as high obstructive dyspnea when the obstructing lesion occurs in the pharynx, larynx or upper trachea; (2) low obstructive dyspnea when it occurs in the lower trachea and main bronchi; (3) bronchial obstructive dyspnea when the obstruction is in the small bronchi. Obstructive dyspnea might also with propriety be called surgical dyspnea because in the majority of instances relief and cure of the condition are obtained only by surgical measures. As an example of the common causes of high obstruction producing dyspnea may be mentioned retropharyngeal abscess, edema of the larynx, foreign body in the larynx or pharynx, laryngeal diphtheria, tumors of the larynx, etc. In high obstruction relief of dyspnea is obtained by surgical removal of the obstruction, intubation or tracheotomy, and while medical assistance is also necessary, as in the laryngeal diphtheria, the emergency requires surgical aid. In low obstruction relief may be obtained by the bronchoscopic removal of the foreign body, by removal of intrabronchial tumors bronchoscopically and by the surgical removal of mediastinal tumors which may be producing tracheal or bronchial compression. In bronchial obstruction dyspnea, where a foreign body or secretion is the etiologic factor, bronchoscopic removal is the surgical method of choice. Spasm plays practically no part in obstructive dyspnea, and for this reason morphine is contraindicated. If the patient is so dyspneic that he cannot sleep it is because he has to use his accessory respiratory muscles to breathe. Sedative doses of morphine will in many instances put him to sleep, stop the action of the accessory respiratory muscles and produce asphyxiation.

Diagnosis. Because of the necessity for surgical aid diagnosis should be made without delay. Careful examination, general and local, and roentgen-ray study is necessary. Laryngoscopy, direct laryngoscopy and bronchoscopy definitely localize the obstruction.

High Obstructive Dyspnea. The symptoms and signs of high obstruction (obstructive laryngeal dyspnea) are pathognomonic. The sign of most importance is the characteristic indrawing of the suprasternal notch, the epigastrium and the intercostal spaces and supraclavicular spaces on inspiration accompanied by dyspnea. The obstruction is greatest on inspiration because of the trapdoor-like action of the vocal cords. The floor of the ventricles of the larynx is higher at the margin of the cord than it is at the outer side of the ventricle. The air passing inward has a tendency to pull the cords together, narrowing the glottis. This produces an increase in the negative pressure on the inside of the chest with the resultant typical indrawing. The sign appears early, particularly in children, is worse on exertion and becomes progressively more marked as the obstruction increases. Usually stridor and wheeze are present. Restlessness in children is a very important sign and inability to sleep, combined with indrawing, is an indication for surgical relief of the dyspnea. The diagnosis is made by the signs and the local examination. The exact condition can best be determined in most cases by direct laryngoscopy and bronchoscopy. If relief cannot be obtained by local endoscopic treatment of the larynx or pharynx intubation may be done or the bronchoscope can be inserted and tracheotomy can be performed properly in an orderly manner with the bronchoscope in position.

Differential Diagnosis. The indrawing and obstructive dyspnea must be distinguished from the indrawing in certain patients with the so-called "soft chest" and in congenital types of chest where the indrawing occurs because of lack of support in the bony and cartilaginous framework. In these cases inspiratory indrawing may occur both at the tip of the ensiform and the suprasternal notch without any sign of dyspnea. There is no wheeze, no stridor, no dyspnea, and bronchoscopic examination is entirely negative for obstruction in the pharynx, larynx, trachea or bronchi.

Low Obstructive Dyspnea. In low obstruction the symptoms and signs are usually characteristic also. The examination of the chest shows indrawing to be greatest at the tip of the sternum and the lower intercostal spaces. There will be little or no indrawing at the

suprasternal notch or above the clavicle. Stridor is usually absent. There is, however, a distinct wheeze which is usually loudest on expiration. The voice is likely to be clear. Roentgenograms may give definite localization of the lesion. The final diagnosis is by bronchoscopic inspection of the lower trachea and bronchi. The treatment is surgical removal of the obstruction, for example, by endoscopic removal of foreign body.

Bronchial Obstructive Dyspnea. In the third type, bronchial obstructive dyspnea, where the obstruction is in the smaller bronchi, there is no indrawing. The clinical picture is very close to that of asthma. There is bilateral emphysema, with an expiratory wheeze. In the cases observed morphine and adrenalin failed to relieve the dyspnea. On bronchoscopic examination, obstructive material was removed from the smaller bronchi with immediate relief of the symptoms.

Discussion. Dr. Joseph F. Stokes: Were it not for the endoscopists, pediatricians would be lost in the handling of these cases. One medical type may be confused with these cases. Intracranial conditions, such as encephalitis, tuberculous meningitis, poliomyelitis, etc., must be distinguished as such and not confused with these obstructive conditions. Such intracranial conditions frequently give paresis of the vocal cords and involve muscles whose action may confuse us with actual obstruction due to a foreign body. In scarlet fever convulsions over a long enough period of time give anoxemia and may not be recognized as an actual cerebral condition. Endoscopy rules out foreign body or some definite form of obstruction but we must remember that in the poliomyelitis type a respirator should be at hand and atropine not withheld. Retropharyngeal abscess may act as a foreign body. Casts in acute bronchopneumonia may be observed far down in the bronchial tree.

PROCEEDINGS

OF THE

SECTION ON GENERAL MEDICINE

PAPERS READ DURING 1931

JANUARY 26

Dr. Francis C. Wood (by invitation) and Dr. E. L. Eliason, "Rheumatic Peritonitis."

Dr. Joseph V. Klauder and Dr. Malcolm J. Harkins (by invitation), "Studies of Human Strains of Bacillus of Swine Erysipelas (Erysipeloid of Rosenbach)."

Dr. Stanley F. Harris (by invitation) and Dr. David Riesman, "Juvenile Hypertension."

Dr. A. M. Ornsteen (by invitation), "Three Cases of Laurence-Biedl Syndrome."

Dr. J. W. Cutler (by invitation) and Dr. J. A. Langbord (by invitation), "The Treatment of Pulmonary Tuberculosis by Artificial Pneumothorax in General Hospitals."

FEBRUARY 23

Dr. William S. Magee (by invitation), "Renal Glycosuria."

Dr. William D. Stroud, Dr. John A. Reisinger (by invitation) and Dr. Louis B. LaPlace (by invitation), "Further Observations Upon 260 Patients with Fibrillating Auricles Over a Period of Two to Eight Years."

Dr. David Cooper and Dr. G. H. Klink, Jr. (by invitation), "Acute Gonococcus Endocarditis."

Dr. William Bates (by invitation), "The Effect of Posture Upon the Position of Abdominal Viscera.

Dr. C. W. Nissler (by invitation), Dr. Eu Shui Tai (by invitation) and Dr. Burgess Gordon, "Some Considerations of the Etiology and Treatment of Obesity."

MARCH 23

Dr. Joseph C. Yaskin (by invitation), "Nervous Symptoms as the Earliest Manifestations of Carcinoma of the Pancreas."

Dr. Louis H. Clerf, "Is Bronchoscopy Indicated in Tuberculosis?"

Dr. V. H. Moon, "Medical Shock: Pathogenesis and Analogy to Surgical Shock."

Dr. Joseph Edeiken (by invitation), "Cardiac Disorders as a Result of Chest Deformity."

Dr. Lewis H. Hitzrot (by invitation) and Dr. Eugene P. Pendergrass, "Intravenous *versus* Oral Cholecystography. A Comparison of the Methods in 100 Patients."

APRIL 27

Dr. Gary Miller (by invitation), "The Prognosis in Pulmonary Tuberculosis."

Dr. Joseph C. Doane, "Some Observations on the Study of a Series of 175 Postmortem Examinations."

Dr. David Riesman, Dr. W. W. Fox (by invitation), Dr. David A. Cooper and Dr. B. J. Alpers (by invitation), "A Case of Hydrophobia Treated by the Pasteur Method, with a Report of Autopsy and Studies of the Central Nervous System.

Dr. William J. Lentz (by invitation), "Rabies in the Dog."

Dr. George Wilson and Dr. Harold Robertson (by invitation), "Facial Diplegia Occurring in Multiple Neuritis."

MAY 25

Dr. John H. Arnett, Dr. Stanley Harris (by invitation), "Electrocardiographic Changes in Pneumonia and Their Implications as Regards Digitalis Therapy."

Dr. Franklin A. Weigand (by invitation), "Chronic Ulcerative Colitis."

Dr. Theodore S. Wilder (by invitation), "The Rôle of Salt-intake in Lobar Pneumonia: With a Statement of Clinical Results."

Dr. Alexander Margolies (by invitation), Dr. Charles C. Wolferth, "The Technique of Combined Electrocardiography and Roentgen Kymography of the Heart Border, Characteristic Curves of Cardiac Contraction."

Dr. J. Alexander Clarke, "The Duration of Bronchial Asthma."

OCTOBER 26

Dr. Michael G. Wohl (by invitation), "Metabolic Changes and Treatment of Obesity."

Dr. Lewis H. Hitzrot (by invitation), Dr. Bernard I. Comroe (by invitation), "Functional Hyperparathyroidism. Report of a Case Improved by Partial Parathyroidectomy."

Dr. Edward Weiss, "Fatal Coronary Occlusion Following an Injection of Novocaine."

Dr. Alexander Margolies (by invitation), Dr. Joseph Edeiken (by invitation), Dr. Charles C. Wolferth, "Human Aortic Pulmonic and Auricular Pulse Curves Recorded by Roentgen Kymography."

Dr. B. B. Vincent Lyon, "Report of a Case of Typhoid Hepatitis. A 'Carrier' of Twenty-six Years' Duration; Cured in Two Years, With a Five-year Follow-up."

NOVEMBER 23

Dr. Stanley E. Harris (by invitation) and Dr. David Riesman, "Carcinoma of the Pancreas with Friedländer Septicemia and Secondary Abscesses of the Liver and Pancreas."

Dr. Harold W. Jones and Dr. Leonardo Tocantins (by invitation), "Infection as an Etiological Factor in Purpura Hemorrhagica."

Dr. P. S. Pelouze, "The Medical Importance of Focal Infective Prostatitis."

Dr. Joseph V. Klauder, "Meiniche's New Clarification Reaction and Müller's 'Ballung's' Reaction in the Diagnosis of Syphilis."

DECEMBER 28

Dr. George C. Griffith (by invitation), "A Case of Pronounced Agranulocytosis with Recovery."

Dr. Thomas Fitz-Hugh, Jr., "Agranulocytic Angina (Pernicious Leukopenia) with Myeloid Cell Hyperplasia of the Bone Marrow."

Dr. W. F. Manges, "Roentgen Ray Diagnosis of Primary Cancer of the Lungs."

Dr. Charles C. Wolferth and Dr. Francis C. Wood (by invitation), "The Importance of Direct Chest Leads in the Diagnosis of Recent Coronary Occlusion."

Dr. Harry Shay, Dr. Albert Katz and Dr. E. M. Schloss (all by invitation), "Experimental Studies in Gastric Physiology. The Evaluation of the Rôle of Duodenal Regurgitation in the Control of Gastric Acidity." (Boldyreff Theory.)

PROCEEDINGS

OF THE

SECTION ON MEDICAL HISTORY

PAPERS READ DURING 1931

FEBRUARY 9

Dr. Thomas R. Currie (by invitation), "The Discoverer of the Trypanosoma."

Dr. W. R. Morse, West China Union University (by invitation), "The Principles Underlying Chinese Medical Practice."

Dr. Joseph Leidy, "American Science in the Fourth Decade of the Nineteenth Century, with Particular Reference to the Beginnings of Modern Biology."

MARCH 9

Professor Charles H. La Wall (by invitation), "The Romance of Quinine."

Dr. Harry Fridenwald, Baltimore, Md. (by invitation), "Certain Portuguese Jewish Physicians: A Romantic Chapter of Medical History."

Dr. Victor Robinson, New York City (by invitation), "On Medical Satire and Caricature."

APRIL 13

Dr. Irving S. Cutter, Chicago (by invitation), "Side Lights on the Etiology of Puerperal Fever."

Dr. C. N. B. Camac, New York City (by invitation), "The Serpent in Medicine."

Dr. Dean Lewis, Baltimore, Md. (by invitation,) "The History and Development of the Surgical Clinic."

NOVEMBER 9

PROGRAM AND EXHIBIT IN HONOR OF SCHAUDINN'S EPOCH-MAKING DISCOVERY.

Dr. John A. Kolmer, Dr. Eugene Case and Dr. George W. Raiziss: An all-day exhibit to cover the main developments in the Diagnosis, Pathology and Treatment of Syphilis from the Historical Standpoint.

Dr. E. B. Kraumbhaar: An exhibition of books, engravings, etc., pertaining to the History of Syphilis.

Dr. Jay F. Schamberg, "Is Syphilis America's Gift to the World?" Professor Henry E. Sigerist, Leipzig (by invitation), "Fracastoro and His Poem."

Dr. John H. Stokes, "Schaudinn."

DECEMBER 14

Dr. Abraham Flexner, New York City (by invitation), "The Graduate School and Full Time Teaching."

ANNUAL REPORT OF THE LIBRARY COMMITTEE FOR 1931

MR. PRESIDENT: In accordance with the ordinances and By-Laws of the College, I herewith submit the following report of the Library Committee for the year 1931:

Total number of volumes in the Library, including the bound volumes and 27,602 unbound "Reports" and "Transactions"	166,706
Number of unbound "Theses" and "Dissertations"	23,921
Number of unbound Pamphlets	202,908
Manuscripts	440

Included in the above are 4463 volumes known as "Reserves," consisting of second copies of some of the more important periodical publications; also 2988 volumes more or less incomplete.

The duplicates, which numer 7832 volumes at this date, are not included in the above total.

The following table shows the number of volumes in the various divisions of the Library:

	Bound.	Incomplete and unbound.	Total.
General Library	122,034	2,988	125,022
Lewis Library	13,855	44	13,899
On permanent deposit:			
S. D. Gross Library	3,781	3	3,784
Library of the Obstetrical Society of Philadelphia	217	0	217
Mütter Museum*	190	0	190
			143,112

Received during the year from all sources, 3829 volumes, 35,962 pamphlets and 12,440 numbers of various periodicals.

* The books of the Mütter Museum are not permitted to be removed from the College building.

Divided as follows:

	Volumes.	Pamphlets.	Journals.
General Library	3,829	35,962	12,440
Lewis Library	5		
S. D. Gross Library	3		
By purchase, General Account	856		
In exchange	679	725	290
	5,372	36,687	12,730

Accessions (including 2 volumes of "Reserves"), 3136.

	Volumes.
General Library	3,128
Lewis Library	5
S. D. Gross Library	3
	3,136

Total increase in the number of volumes for the year, 3136.

Photographs received in response to requests sent out during the past year and voluntary gifts, 15. Fellows of the College, 0; other than Fellows, 15.

Total number of portraits listed to date, 22,622.

The individual "Donors" for the year ending November 1, 1931, number 527; this represents 868 distinct presentations. Each gift is duly acknowledged.

The following list shows individual gifts of twenty volumes and over; also the number of volumes presented by the various publishing houses:

	Volumes.
Dr. A. P. C. Ashhurst	24
Dr. Charles Baum	123
Dr. J. C. Gittings	240
Dr. E. B. Krumbhaar	24
Dr. G. C. Kuesel	32
Dr. Charles K. Mills	156
Dr. James Thorington	29
Dr. P. F. Williams	41

From the publishing houses of:

	Volumes.
P. Blakiston's Son & Co.	35
F. A. Davis Company	20
Lea & Febiger	17
J. B. Lippincott Company	18
C. V. Mosby Company	1
W. B. Saunders Company	67

The library is indebted for large gifts of pamphlets and unbound periodicals to the following donors:

Dr. H. B. Allyn
Dr. J. H. Arnett
Dr. A. P. C. Ashhurst
Dr. John Aulde
Dr. Charles Baum
Dr. H. L. Bockus
Dr. C. W. Burr
Dr. Burton Chance
Dr. J. Solis Cohen
Dr. F. X. Dercum
Dr. W. A. N. Dorland
Dr. G. M. Dorrance
Dr. A. A. Eshner
Dr. H. M. Fisher
Dr. L. F. Flick
Dr. J. P. C. Griffith
Dr. S. Mc C. Hamill
Dr. H. A. Hare
Mr. Paul B. Hoeber
Dr. R. J. Hunter
Dr. J. H. Jopson
Dr. W. W. Keen
Dr. E. B. Krumbhaar
Dr. G. C. Kuesel
Dr. Joseph Leidy
Dr. C. K. Mills
Dr. S. W. Moorhead
Dr. C. C. Norris
Dr. G. W. Norris
Philadelphia Pathological Society
Rockefeller Foundation
Dr. G. E. deSchweinitz
Dr. E. A. Shumway
Dr. James E. Talley
Dr. James Thorington
U. S. Department of Agriculture
U. S. Department of Commerce
U. S. Department of Interior
U. S. Public Health Service
University of Pennsylvania, Graduate School of Medicine
Dr. Joseph Walsh
Dr. P. F. Williams
Dr. J. C. Wilson
Dr. C. S. Witherstine
Dr. William Zentmayer

684 new publications were added to the Library during the past year, viz., United States, 315; Great Britain and Colonies, 52; France, 192; Germany, 190; Italy, 11; all other countries, 31. Of these new publications, 27 were written or edited by Fellows of the College.

21 volumes were presented by the following authors or editors:

Dr. K. E. Appel
Dr. A. P. C. Ashhurst
Dr. L. F. Baker
Dr. A. A. Cairns
Dr. G. M. Dorrance
Dr. P. L. Drouet
Dr. H. Friedenwald
Dr. J. Hamel
Dr. A. E. Hertzler
Captain R. C. Holcomb, M.D.
Dr. C. F. Krafft
Dr. C. Leventis
Dr. Thomas McCrae
Dr. C. C. Miller
Dr. N. Mumey
Dr. C. C. Norris
Dr. L. H. Roddis
Dr. F. A. Stahl
Dr. E. A. Strecker
Dr. James J. Walsh
Dr. W. H. Wilmer

14 volumes were sent by the publishers at the request of the following authors or editors:

Dr. A. C. Buckley	Dr. S. A. Loewenberg
Dr. A. E. Bulson	Dr. W. E. MeVey
Dr. J. C. DaCosta	Dr. P. S. Pelouze
Dr. C. B. Drake	Dr. A. A. Stevens
Dr. F. G. Ebaugh	Dr. F. T. Stewart
Dr. H. A. Hare	Dr. P. F. Williams
Dr. W. E. Lee	Dr. J. K. Young

Summary of the "Funds":

	Volumes purchased.	Cost.
Henrietta Rush Fales Baker Fund	27	$231.38
Luther S. Bent Fund	8	22.66
William T. Carter Fund	8	38.66
Gerardus Clarkson Fund	1	7.13
Francis X. Dercum Fund	24	145.45
Louis A. Duhring Fund	25	321.57
Augustus A. Eshner Fund	46	252.51
John D. Griscom Fund	50	177.18
Richard H. Harte Fund	41	345.98
Charles T. Hunter Fund	1	
William F. Jenks Fund	54	278.13
Oliver A. Judson Fund	6	16.21
William V. and John M. Keating Fund	11	37.32
William W. Keen Fund	50	229.67
Henry Leffman Fund	0	00
Library Endowment Fund	205	760.54
Horace Magee Memorial Fund	99	547.40
J. Ewing Mears Fund	32	139.67
Charles K. Mills Fund	1	6.11
Weir Mitchell Fund	76	209.05
E. E. Montgomery Fund	23	88.31
John H. Musser Fund	0	00
Elizabeth K. Newcomet Fund	5	18.03
William F. Norris Fund	25	202.10
Charles A. Oliver Fund	8	19.02
Philadelphia Medical Society Fund	8	9.05
Lewis Rodman Fund	18	146.27
Douglas Stockton Warren Fund	18	57.61
John F. Weightman Fund	5	28.29
J. William White Fund	30	176.19
Caspar Wistar Fund	40	126.45
S. Lewis Ziegler Fund	6	20.26
	951	$4,658.20
From the General Account—by purchase	856	$6,480.84

Special accounts:

	Volumes purchased.	Cost.
Fund for completing files of journals	60	$288.13
Fund for rare and valuable books	23	823.18
S. D. Gross Library Account	9	00

	Expended.
Catalogue Endowment Fund—on account of salaries	$46.50
Louis A. Duhring Fund—for general purposes of the Library	11,013.00
Morris Longstreth Fund—on account of salaries	1,105.81
George B. Wood Fund—Library supplies, stationery, etc.	364.38

Number of volumes bound, 1863.

	Visitors.	Fellows.	Total.
Use of Library	14,416	2,025	16,441

The Library has been kept open two evenings each week and on the five minor legal holidays for the same period of time as during the previous year:

Evenings.	Visitors.	Fellows.	Total.
74	1,325	149	1,474

Legal holidays.	Visitors.	Fellows.	Total.
5	101	29	130

These figures are included in the total given for the year.

	1931.	1930.	Increase.
Number of volumes consulted in the Library	63,296	49,244	14,052

The number of books "consulted in the Library" includes only those supplied on demand. Readers have access to the bound volumes of periodicals and works of reference kept on the shelves in the Reading Room, and the Fellows of the College have access to the Book Stacks. There are, therefore, many volumes consulted of which no accurate record can be kept.

	1931.	1930.	Increase.
Number of volumes taken out	4,480	3,708	772

Use of Study Rooms: Average number of rooms in use throughout the year, 3.

Number of volumes in these rooms during the year, 1164.

	Works.	Volumes.	Cards written.	Printed cards revised.
Cataloguing	738	750	8,618	1,282

All books added to the shelves during the past year and 155 of the more important pamphlets have been catalogued and shelf-listed; and all bound volumes, including periodicals, transactions and reports, have been accessioned.

Revision of the Catalogue: 11,701 cards have been revised, rewritten and filed.

2841 unbound pamphlets and reprints have been subject-headed, and arranged alphabetically by subject and by author under the subject.

We have listed November 1, 1931, 1455 current periodical publications, including "Transactions" and "Reports," obtained through the following sources:

	American.	Foreign.
Endowment Fund	52	69
By purchase from General Account	173	380
In exchange	105	124
Editors	193	39
Publishers	28	3
Gifts from various Donors	118	43
	669	658
Reports not included in the above	85	43
	754	701

The actual number of periodicals received during the year, including new subscriptions (5 American, 11 Foreign), total 1327.

The following is a list of the foreign schools of medicine with which we exchange publications at the present time:

University of Amsterdam	University of Kiel
" Basel	" Königsberg
" Berlin	" Lausanne
" Bonn	" Liege
" Breslau	" Lund
" Erlangen	" Marburg
" Giessen	" Paris
" Göttingen	" Würzburg
" Heidelberg	

During the past year we have heard from all of the above. 1103 dissertations and 842 theses have been added to our collection.

We have received in cash from the sale of duplicates for the current year, $151.10.

We have distributed books and journals on exchange account to the following:

Boston Medical Library, Boston, Massachusetts
Howard University, School of Medicine Library, Washington, D. C.
New England Deaconess Hospital, Medical Library, Boston, Massachusetts
University of Maryland, Medical Department, Baltimore, Maryland
University of Pennsylvania, Medical Department, Philadelphia

We have received exchanges from:

Academy of Medicine of Northern New Jersey, Newark, New Jersey
Boston Medical Library, Boston, Massachusetts
Detroit Public Library, Medical Service Department, Detroit, Michigan
Free Public Library, Trenton, New Jersey
Harvard University, School of Medicine and Public Health Library, Boston, Massachusetts
Jackson County Medical Society, Kansas City, Missouri
McGill Medical Library, Montreal, Canada
Medical Society of the County of Kings, Library, Brooklyn, New York
New York Academy of Medicine Library, New York City
New York Post Graduate Medical School, New York City
Prentiss Library, New York Homœpathic Medical and Flower Hospital, New York City
Public Library, Plainfield, New Jersey
St. Luke's Hospital Medical Library, Chicago, Illinois
University of Alabama, School of Medicine, Library, Alabama
University of California, Medical School, Library, San Francisco, California
University of California, Zoölogical Laboratory, Berkeley, California
University of Michigan, General Library, Ann Arbor, Michigan
University of Nebraska, College of Medicine, Library, Omaha, Nebraska
University of Oregon, Medical School, Library, Portland, Oregon
University of Pennsylvania, School of Medicine, Philadelphia
University of Texas, School of Medicine, Library, Galveston, Texas
University of Western Ontario, Medical School, Library, London, Ontario
Washington University, School of Medicine, St. Louis, Missouri

A number of the exchanges on the above list were made through the Medical Library Association.

With the aid from exchanges and purchases made with the funds

appropriated by the Library Committee, we have been able to complete the files of the following journals:

Congrès des Médecins, Aliénistes et Neurologistes
Ergebnisse der Chirurgie und Orthopaedie
Internationales Centralblatt für Laryngologie
Journal of the Allied Dental Societies
Journal of the New York Institute of Stomatology
Medicina Contemporanea

In reponse to requests sent out during the year we have received acceptances from 35 Foreign and 33 American publishers and editors to send their periodical publications as issued, either as a gift or in exchange for the Transactions of the College.

Amount of fines collected for the current year ending November 1, 1931, $41.25.

List of rare medical books and works of special interest received during the year. (These rare books have been acquired without in any way interfering with the purchase of current medical publications):

INCUNABULA

[Total number of incunabula at this date 400 Titles, 402 Volumes.]

Aquino, Thomas de Ord. Praedic. Opuscula. Venetiis, Hermannus Lichtensteyn, Coloniensis, 1490.

Fund for Rare Books.

Avicenna. Metaphysica sive ejus prima philosophia.*** Venetiis, Hieronymus de Durantis per Bernandinum de Vitalibus, 1495. [Hain No. 2217. Gesamtkatalog No. 3130.]

Presented by the Boston Medical Library.

Brack, Wenceslaus. Vocabularium Archonium nuncupatum.*** [Argentorati, Johannes Reynardi alias Grüninger, circa 1486.] [Hain-Copinger No. 3697.]

Presented by "The Friends of the College Library."

Cherubini da Spoleto, Ordin. Minor. Regola della vita spirituale e matrimoniale.*** [Florentiae, Franciscus de Dino, Jacobi Chartarii fil., circa 1487.] [Hain No. 4930.]

Presented by "The Friends of the College Library."

Duns, Johannes, Scotus, Ordin. Minor. Elenchi seu Quaestiones super libros Elenchorum Aristotelis.*** Venetiis, Johannes de Landovia, dictus Hertzog, 1499. [Hain No. 6439.] [Bound with his: Quaestiones in metaphysicam Aristotelis. 1499.]

Presented by "The Friends of the College Library."

Duns, Johannes, Scotus, Ordin. Minor. Quaestiones in metaphysicam Aristotelis.*** Venetiis, Johannes de Landovia, dictus Hertzog, 1499. [F. 1 missing.] [Hain No. 6451.] [Bound in old vellum manuscript.] [Contains also his: Elenchi seu Quaestiones super libros Elenchorum Aristotelis. 1499.]

Presented by "The Friends of the College Library."

Platea, Franciscus de Ordin. Minor. Opus restitutionum, usurarum et excommunicationum. Venetiis, Johannes de Colonia et Johannes Manthen de Gherretzem, 1477. [Contemporary manuscript notes.] [Rubricated initials and paragraph marks.]

Fund for Rare Books.

Schellig, Conradus. [1450–1510] [In morbum gallicum.] Heidelbergae, Fridericus Misch, 1495–6. [Reichling II No. 727.] [First printed book on syphilis. Sudhoff.]

Fund for Rare Books.

Themistius. Paraphrasis in Aristotelis.*** Venetiis, Octavianus Scotus, Modoetiensis, per Bartholomeum de Zanis de Portesio, 1499. [Hain No. 15464.]

Presented by "The Friends of the College Library."

Works of Special Interest

Agr[ippa], Henricus Cornelius. Paradoxe svr l'incertitvde, vanite, et abus des sciences. Traduite en Francois, du Latin. [Antverpiae, Graphaeus], 1603.

Fund for Rare Books.

Aristoteles. Libri qvi vvlgo parua naturalis à philosophie appellantur.*** Parisiis, Ex officina Prigentii Caluarini ad Geminas Cyppas in Clauso Brunello, 1541.

Fund for Rare Books.

Aurelianus, Caelius. Medici vetusti, et tractanda morborum curatione diligentissimi secta methodici.*** Lvgdvni, apvd Gvliel. Rovillivm, 1566.

Fund for Rare Books.

Barbette, Paul. Oevvres chirvrgiqves et anatomiqves,*** appropriées à la circulation du sang et autres découvertes des modernes; avec vn traité de la peste enrichi d'observations. Lyon, Faeton, 1680.

Fund for Rare Books.

[Begon, Etienne]. Recueil general, des pieces contenues av procez de Monsieur le marquis de Gesures, et de Mademoiselle de Mascranni, son epouse. Nouvelle éd. 2 v. Rotterdam, Leers, 1714.

Fund for Rare Books.

Cocchi, Raimondo, Lezioni fisico-anatomiche recitate pubblicamente in Firenze nel teatro del regio spedale di Santa Maria nuova.*** Livorno, Masi, 1775.

Fund for Rare Books.

Elsholtz, Joannes Sigismundus. Anthropometria, sive de mutua membrorum corporis humani proportione, et naevorum harmonia libellus. Editio post Patavianam altera, figuris aneis illustrata. Apud Rupertum Volckern. Francofurti ad Oderam, Beemani, 1663.

Fund for Rare Books.

Faithful and full account of the surprising life and adventures of the celebrated doctor Sartorius Sinegradibus. As also of the many wonderful operations he performed in this city. Edinburgh, for the Author, [1700].

Presented by Burton Chance, M.D.

Faithful and full account of the surprising life and adventures of the celebrated Dr. Sartorius Sinegradibus. As also of the many wonderful operations he performed in this city. London, Exeter exchange, 1749. [Extr.—from the works of the celebrated Rabelais.]

Presented by Burton Chance, M.D.

Galen, [Claudius]. De plenitudine liber. Jo. Guinterio Andernaco interprete. Paris, Wechel, 1531.

Presented by Charles W. Burr, M.D.

Galen, Claudius. [Opuscula]. De elementis libri duo Victore Trincavelio interprete. De naturalibus facultatibus libri tres. Thoma Linero interprete. Acc. Iac. Sylvii scholia.

De diebus decretoriis libri tres. Io. Guinterio Andernaco interprete. The three works in the one volume. Lvgdvni, Rouillius, 1558.

Presented by "The Friends of the College Library."

Gamet, J. M. Théorie nouvelle sur les maladies cancéreuses, nerveuses et autres affections du meme genre, avec des observations, pratiques sur les effets de leur remede approprié. 2 v. in 1. Paris, Ruault, 1772.

Fund for Rare Books.

Goulard, [Thomas]. Traité sur les effets des preparations de plomb, et principalement de l'extrait de saturne, employé sous differentes formes, et pour differentes maladies chirurgicales. 2 v. in 1. Pezenas, Fuzier, 1760.

Fund for Rare Books.

Illustrium Hollandiae et Westfrisiae ordinum alma Academia Leidensis. Lvgdvni Batavorum, apud Iacobum Marci, & Iustum à Colster, 1614.

Fund for Rare Books.

Keill, [James]. Tentamina medico- physica.*** Medicina statica britannica.*** Editio novi. Lugduni Batavorum, apud Joannem Arnold Langerak, 1730.

Fund for Rare Books.

de La Condamine, [Charles Marie]. Discourse on inoculation. Read before the Royal Academy of sciences at Paris, the 24th of April, 1755. London, Vaillant, 1755.

Fund for Rare Books.

Lieutaud, [Joseph]. Anatomie historique et pratique. Augmentée*** par [Antoine] Portal. Nouvelle édition. 2 v. Paris, Vincent, 1776.

Fund for Rare Books.

Lieutaud, [Joseph]. Precis de la matiere medicale. Traduction de la seconde partie du Précis de la médecine pratique, publié en Latin. v. 2. Paris, Vincent, 1766.

Fund for Rare Books.

Mercurialis, Hieronymus. Medicina practica, seu de cognescendis, discernendis et curandis omnibus humani corporis affectibus, earumque causis indagandis, Libri V.*** in lucem, studio et opera, Petri de Spina. Postrema editio. Lvgdvni, Pillehotte, 1623.

Fund for Rare Books.

Morello, Francisco. Medicinale patrocinium in sanguinis circulationem. Neapoli, Ex officina Bulifoniana superiorum permissu, 1678.

Fund for Rare Books.

Navier, Pierre Toussaint. Réflexions sur les dangers des exhumations précipités, et sur les abus des inhumations dans les églisés suivies d'observations sur les plantations d'arbres dans les cimetieres. Amsterdam, Morin, 1775.

Fund for Rare Books.

Pascoli, Alessandro. Corpo-umano, o breve storia.*** Venezia, Poletti, 1727.

Fund for Rare Books.

Paulus Aegineta. Totius rei medicae libri VII. ad profectionem parati, et brevi summa omnem artem complectentes. Per Ianum Cornarium medicu[m] physicum latina lingua co[n]scripti.*** Basileae, Hervagius, 1556. [Contains also—Bock, II. Kreüter Buch.*** 1560.]

Presented by Charles W. Burr, M.D.

Petit-Radel, Ph[ilippe]. Institutions de médecine, ou exposé sur la théorie et la pratique de cette science, d'après les auteurs anciens et modernes, ouvrage didactique contenant les connaissances générales, nécessaires à ceux qui se destinent à exercer l'art de guérir. Paris, Agasse, an IX [1802].

Fund for Rare Books.

A Physician. Advice to the physician, the surgeon, and apothecary, and to their patients; after the manner of Dean Swift; with notes practical and illustrative, founded on long observation that may be beneficial to them all, and favorable to health and long life. London, Fearman, 182[1].

Fund for Rare Books.

Physician. A parallel between the late celebrated Mr. Pope, and Dr. Taylor, oculist to the king of Great Britain.*** 4 ed. [London], Printed for the Author, 1749.

Presented by Burton Chance, M.D.

Placet, Francois. La superstition du temps, reconnue aux talismans, figures astrales, et statuës fatales.*** Paris, Alliot, 1667. [Contains also his—Poudre de sympathie soupconnée de magie.]

Fund for Rare Books.

Salernum, School of. Schola Salernitana, sive de conservanda valetudine precepta metrica. Autore Joanne de Mediolano hactenus ignoti. Cum luculenta et succincta Arnoldi Villanovani in singula capita et exegesi. Ex recensione Zachariae Sylvii.*** Nova editio.*** Ratisponae, Seidelius, 1711.

Fund for Rare Books.

Sennert, Daniel. De febribus libri IV. Cui accessit fasciculus medicamentorum contra pestem. 3 ed. Parisiis, apud Societatem, 1633.

Fund for Rare Books.

Short, Thomas. General treatise on the different sorts of cold mineral waters in England, with a variety of experiments for examining and ascertaining their virtues and uses, and discovering their effects on the blood and juices of the human body.*** London, printed for the Author, 1766.

Fund for Rare Books.

Thelwall, John, Esq. Letter to Henry Cline, Esq. On imperfect developments of the faculties, mental and moral, as well as constitutional and organic; and on the treatment of impediments of speech. London, Taylor, 1810.

Presentation copy from the author to Dr. William Lister, and with the library ticket of the latter.

Fund for Rare Books.

Tissot, [Samuel] A[uguste André David]. Essay on diseases incident to literary and sedentary persons, with proper rules for preventing their fatal consequences, and instructions for their cure. With preface and notes by J. Kirkpatrick.

London, Nourse, 1769.

Formerly the property of Benjamin Smith Barton.

Presented by A. P. C. Ashhurst, M.D.

Manuscripts

Norris, George Washington. Varioloid and vaccine diseases. [Phila., 1830.] [Manuscript,] Theses, University of Pennsylvania, Medical department.

Presented by G. W. Norris, M.D.

Norris, George William. Moors in Spain. [Phila., 1895.] [Typewritten manuscript.] [Bound with—Norris, G. W. Varioloid and vaccine diseases, 1830.] Theses, University of Pennsylvania, Senior Arts.

Presented by G. W. Norris, M.D.

Norris, William Fisher. Generation and development. [Phila., 1861.] [Manuscript,] Theses, University of Pennsylvania, Medical department. [Bound with—Norris, G. W. Varioloid and vaccine diseases. 1830.]

Presented by G. W. Norris, M.D.

Warthin, Aldred Scott. On the occurrence of numerous large giant cells in the tonsils and pharyngeal mucosa in the prodromal stage of measles. [Ann. Arbor, 1931.] [Manuscript.]

Presented by C. V. Weller, M.D. through the courtesy of E. B. Krumbhaar, M.D.

Other Interesting Additions

Bronze Medal inscribed "To Nicholas Senn, the Master Surgeon; Memorial from his fellows, November 11, 1905." [Presented to Dr. Ashhurst by William N. Senn, Chicago, Ill., September 25th, 1907.]

Presented by A. P. C. Ashhurst, M.D.

We have also received during the past year a number of interesting and more or less valuable autograph letters.

Use of the Photostat: During the past year there have been furnished to Fellows of the College 507 prints, and to other than Fellows, on application, 357 prints. These outside applications include requests from Denver, Col.; Wichita, Kansas; New York, Wisconsin, and Philadelphia, Pa.

The work for the Library has been continued as heretofore according to the instructions of the Library Committee, 2449 large and small prints have been made; 10 volumes of incunabula completed as well as various portraits and manuscripts.

Up to the present time there have been made photostat reproductions of 293 incunabula.

There has been an increase to the income of the Library Endowment Funds through the gift of Dr. Henry Leffman, who left a fund of $62,000, in trust with the Fidelity Philadelphia Trust Company. One fifth of the income of this fund to be paid to the Library of the College of Physicians of Philadelphia.

A complete list of all the Funds and the expenditures for the year is given in a previous part of this report. All additional expenses, salaries, binding, etc., have to be governed according to the funds annually appropriated by the College to the Library Committee.

An account of stock has been taken and any displacements found arranged in their proper order; no report has been made of any book missing from the shelves.

The Library Committee discussed the advisability of obtaining a man as first assistant in the Library who, acquiring knowledge of the various details, would be able to fill the office of Librarian when it became vacant.

LIBRARY COMMITTEE

GEORGE W. NORRIS, M.D.,
Chairman (Resigned).

ALBERT P. BRUBAKER, M.D.,
Chairman.

INDEX

A

Activity, sympathetic, in certain diseases, 30
Alimentary and urinary tracts, diverticulosis of, 182
Annual report of Library Committee, 297
Appendix, 201
Austin, J. Harold, therapeutic use and abuse of fluids, 8
and Ravdin, I. S., intravenous therapy in dehydration and toxemia, 10

B

Babbitt, James A., discussion on Ballance-Duel research, 283
on how Dr. G. Hudson Makuen treated stammering, 245
on motion pictures of larynx, 285
on postoperative treatment by radium in radical mastoidectomy, 252
modified attic drainage in chronic suppurative otitis media, 258, 261
Batson, O. V., discussion on lesions of pterygomaxillary fossa, 249, 250
on modified attic drainage in chronic suppurative otitis media, 259
usual course and relations of nervus facialis and practically important variations, 252
Bauer, E. L., discussion on community health program, 83
Beer, Edwin, diverticulosis at alimentary and urinary tracts, 182
Bismuth in treatment of syphilis, 101
Blair, V. P., blepharoplastic and ptosis operative procedure, 228
Bonner, W. F., left plastic iritis which does not respond to mydriatic and parenteral protein injections, 228

C

Carpenter, J. T., and Carpenter, Chapin, nonglaucomatous halos, 219
Circulation, peripheral, sympathetic activity in diseases of, 30
Clerf, Lewis H., discussion on motion pictures of larynx, 286
Coates, George M., discussion on cerebrospinal rhinorrhea, 273
on how Dr. G. Hudson Makuen treated stammering, 245
on modified attic drainage in chronic suppurative otitis media, 260
on motion picture demonstration of radical maxillary sinus operation under local anesthesia, 289
pictures of radical frontal sinus operations, 288
Community health program, 83
Cornell, Walter S., discussion on noise in industry, 267
Frey, Marie Currie, Laird, Miss Roberta, audiometric findings and clinical records in Philadelphia Public Schools, 263

Cowan, Alfred, discussion on melanosis of anterior segment of globe with elevation on iris, 214
and McAndrews, L. F., localizing tears in retinal detachment, 216
Craig, Frank A., discussion on community health program, 87
Crawford, B. L., discussion on lymphomas of conjunctiva, 234
Cross, George H., reconstruction of eye socket, 203
removal of oxidized tissue, remaining after foreign body of cornea (a handle for corneal burrs), 231

D

Davis, Warren B., discussion on cerebrospinal rhinorrhea, 273
on osteoma of paranasal sinuses, 279
Dehydration, intravenous therapy in toxemia and, 10
Dintenfass, Henry, discussion on motion pictures of larynx, 286
motion picture demonstration of radical maxillary sinus operation under local anesthesia (Caldwell-Luc), 288, 289
Diverticulosis of alimentary and urinary tracts, 182
Donnelly, J. C., cerebrospinal rhinorrhea, 271
discussion on modified attic drainage in chronic suppurative otitis media, 260
Duel, Arthur B., short résumé of results of Ballance-Duel research in nerve anastomosis and employment of nerve grafts in surgical treatment of facial palsy, 279, 284

E

Egbert, Seneca, discussion on community health program, 88

F

Fay, Temple, discussion on lesions of pterygomaxillary fossa, 249
Fisher, John M., discussion on community health program, 89
Fluids, therapeutic use and abuse of 8
Frazier, Charles H., discussion on Ballance-Duel research, 282

G

Garner, Vaughn C., newer conception of diagnosis and treatment of early syphilis, 118
Gibbon, John H., discussion of intravenous protein therapy, from a surgical standpoint, 24
on intravenous therapy from a surgical standpoint, 25
Gleason, Edward B., discussion on modified attic drainage in chronic suppurative otitis media, 260
on motion pictures of larynx, 266
of radical frontal-sinus operations, 287
on usual course and relations of nervus facialis and practically important variations, 252

H

Health program, community, 66, 90
Heatley, C. A., motion pictures of larynx, 284, 286
Holloway, T. M., discussion on case of bilateral optic atrophy due to methyl alcohol poisoning, 231
on homonymous hemianopsia in diabetes, 205
sarcoma of choroid, 238
and Cowan, Alfred, spontaneous complete absorption of cataractous lens, 222, 224
and Fry, W. E., unsuspected brawny scleritis in case of retinal detachment with secondary glaucoma, 208, 209
Houser, Carl L., discussion on motion pictures of radical frontal-sinus operations, 288
Hunter, Robert J., discussion on Ballance-Duel research, 283
on osteoma of paranasal sinuses, 279
lesions of pterygomaxillary fossa, 247, 256

I

Intravenous therapy, discussion from a surgical standpoint, 25
symposium on, 8

K

Keeler, Joseph C., postoperative treatment by radium in radical mastoidectomy, 251, 252
Kern, Richard A., nonspecific protein therapy, 17
Klauder, Joseph V., recent experimental contributions to syphilis, 92
and Perce De Long, lupus erythematosus of conjunctiva and eyelids, 232
and Robertson, Harold F., treatment of interstitial keratitis, 206
Kolmer, John A., recent developments in serology of syphilis, 97
Krauss, Frederick, cysts of orbit, 205
large penetrating wound of lower lid and eyeball, 203, 204

L

Lang, F. J., osteoarthritis deformans contrasted with osteoarthritis deformans juvenilis, 144
Langdon, H. Maxwell, melanosis of anterior segment of globe with elevations on iris, 212, 214
Lewis, Fielding O., discussion on early intrinsic cancer of larynx, 257
on malignant conditions of laryngopharynx, 263
on value of roentgen diagnosis in diseases of neck and upper respiratory tract, 242

Lewis, Sir Thomas, vasovagal syncope and carotid sinus mechanism, with comments on Gowers' and Nothnagel's syndromes, 132
Loftus, John E., discussion on cerebrospinal rhinorrhea, 273
London and Edinburgh, how they influenced medicine in Philadelphia in eighteenth century, 151

M

MacFarlan, Douglas, discussion on noise in industry, 266
experimental proof that voice cannot be maintained at a level, 246
McIlvain, Edwin H., discussion on noise in industry, 267
Magrath, George Burgess, medical science in service of state, some professional experiences, 200
Manges, Willis F., discussion on value of roentgen diagnosis in diseases of neck and upper respiratory tract, 242
Medical science in service of state, some professional experiences, 200
Medicine in Philadelphia in eighteenth century, how London and Edinburgh influenced, 151
Memoir of Dr. Joseph Seal Neff, lxv
Moore, Joseph Earle, syphilitic optic atrophies, 226

N

Neff, Dr. Joseph Seal, memoir of, lxv
Nonspecific protein therapy in surgical condition, 21
some cautions as to its use, 17

O

Ornsteen, Abraham, discussion on Ballance-Duel research, 283
Orton, Henry B., malignant conditions of laryngopharynx, 261
Osteoarthritis deformans contracted with osteoarthritis deformans juvenilis, 144

P

Packard, Francis B., annual address of President, 1
how London and Edinburgh influenced medicine in Philadelphia in eighteenth century, 151
Pancoast, H. K., roentgenographic anatomy and physiology of larynx and adjacent structures, 239
Pelouze, P. S., ophthalmological importance of focal infective prostatitis, 236
Pendergrass, E. P., discussion on radiational cataract, 215
value of roentgen diagnosis in diseases of neck and upper respiratory tract, 240

Peripheral circulation, sympathetic activity in certain diseases, especially those of, 30
Peter, Luther C., discussion on localizing tears in retinal detachment, 216
recession operation in squint, 234, 236
Pfeiffer, Damon B., discussion on intravenous protein therapy from a surgical standpoint, 27
Pillsbury, Donald M., prevention and treatment of prenatal syphilis, 122
Posey, W. C., discussion on spontaneous complete absorption of cataractous lens, 224
President, annual address of, 1
Pressman, F. G., discussion on modified attic drainage in chronic suppurative otitis media, 260
Proceedings of Section on General Medicine, 292
on Medical History, 295
on Ophthalmology, 203
on Otology and Laryngology, 239

R

Rademaker, L. A., discussion on intravenous therapy, 29
nonspecific protein therapy in surgical conditions, 21
Randall, B. Alexander, discussion on Ballance-Duel research, 283
on how Dr. G. Hudson Makuen treated stammering, 245
on lesions of pterygomaxillary fossa, 250
Reese, W. S., discussion on case of spontaneous cyst of iris, 211
on recession operation in squint, 236
Ridpath, Robert F., discussion on motion picture demonstration of radical maxillary sinus operation under local anesthesia, 288
of larynx, 286
Rosenberger, Randle C., discussion on community health program, 85

S

Scarlett, Hunter, senile macular changes of retina, associated with focal infection, 226
Schamberg, Jay Frank, fever therapy in treatment of syphilis, 108
Schoch, Arthur G., newer arsenicals in treatment of syphilis, 113
deSchweinitz, G. E., discussion on case of bilateral optic atrophy due to methyl alcohol poisoning, 231
on case of bitemporal paracentral scotoma, 218
on hazards of antifreeze methanol, 221
on melanosis of anterior segment of globe with elevation on iris, 213
on ophthalmological importance of focal infective prostatitis, 237
homonymous hemianopsia in diabetes, 205
and Baer, B. F., note on radiational cataract, 214
Scott, W. J. Merle, and Morton, John J., sympathetic activity in certain diseases, especially those of peripheral circulation, 30

Shannon, C. E., case of bilateral optic atrophy due to methyl alcohol poisoning, 229
Shannon, C. E. G., case of bitemporal paracentral scotoma, 217
and Andrews, L. F., lymphomas of conjunctiva, 233, 234
Shumway, Edward, A., case of spontaneous cyst of iris, 211
discussion on hazards of antifreeze methanol, 221, 222
on radiational cataract, 216
Shuster, B. H., discussion on lesions of pterygomaxillary fossa, 250
of serious complications following tonsillectomy, 278
Skillern, Samuel, discussion on cerebrospinal rhinorrhea, 274
motion pictures of radical frontal sinus operations, 286, 288
Smith, J. Austin, osteoma of paranasal sinuses, 278
Smyth, Henry Field, noise in industry, its effects on hearing and general health, 264
Spiller, William C., discussion on Ballance-Duel research, 283
Stauffer, Nathan P., discussion on motion pictures of larynx, 286
of radical frontal sinus operations, 287
Steel, Mary S., how Dr. G. Hudson Makuen treated stammering, 243, 245
Stokes, John H., discussion on syphilitic optic atrophies, 228
syphilis as a problem in preventive medicine, 127
Stokes, Joseph F., discussion on motion picture demonstration of obstructive dyspnea, 291
Stout, Philip, discussion on experimental proof that voice cannot be maintained at a level, 246
on osteoma of paranasal sinuses, 279
on postoperative treatment by radium in radical mastoidectomy, 252
State, medical science in service of, 200
Sympathetic activity in certain diseases, especially those of peripheral circulation, 30
Symposium, on intravenous therapy, 8
Syphilis, bismuth in treatment of, 101
discussion on, 92
fever therapy in treatment of, 108
newer arsenicals in treatment of, 113
conceptions of diagnosis and treatment of, 118
prenatal, prevention and treatment of, 122
as problem in preventive medicine, 127
recent experimental contribution to, 92
serology of, present developments in, 97

T

Therapeutic use and abuse of fluids, 8
Therapy, intravenous, symposium on, 8
Toxemia, intravenous therapy in dehydration and, 10
Trumper, Max (by invitation), hazards of antifreeze methanol, 220

Tucker, Gabriel, discussion on malignant conditions of laryngopharynx, 263
on value of roentgen diagnosis in diseases of neck and upper respiratory tract, 242
early intrinsic cancer of larynx, 254, 257
motion picture demonstration of obstructive dyspnea, bronchoscopic observations, 289

U

Urinary and alimentary tracts, diverticulosis of, 182

V

Vasovagal syncope and carotid sinus mechanism, with comments on Gowers' and Nothnagel's syndrome, 132

W

Weeder, N. D., discussion on modified attic drainage in chronic suppurative otitis media, 260
Winslow, C.-E. A., community health program, 66, 90
Wood, George B., discussion on cerebrospinal rhinorrhea, 274
indications for removal of tonsils and adenoids, 268
serious complications following tonsillectomy, 274
Wright, Carroll S., bismuth in treatment of syphilis, 101

Z

Zentmayer, William, congenital cararact with unusual visual acuity, 209
discussion on bitemporal paracentral scotoma, 218
on case of bilateral optic atrophy due to methyl alcohol poisoning, 230
on large, penetrating wound of lower lid and eyeball, 204
on lymphomas of conjunctiva, 234
on ophthalmological importance of focal infective prostatitis, 237
on spontaneous complete absorption of cataractous lens, 225